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of
OPHTHALMOLOGY
and
OTOLARYNGOLOGY

FORTY-EIGHTH
ANNUAL MEETING

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October 10-13, 1943

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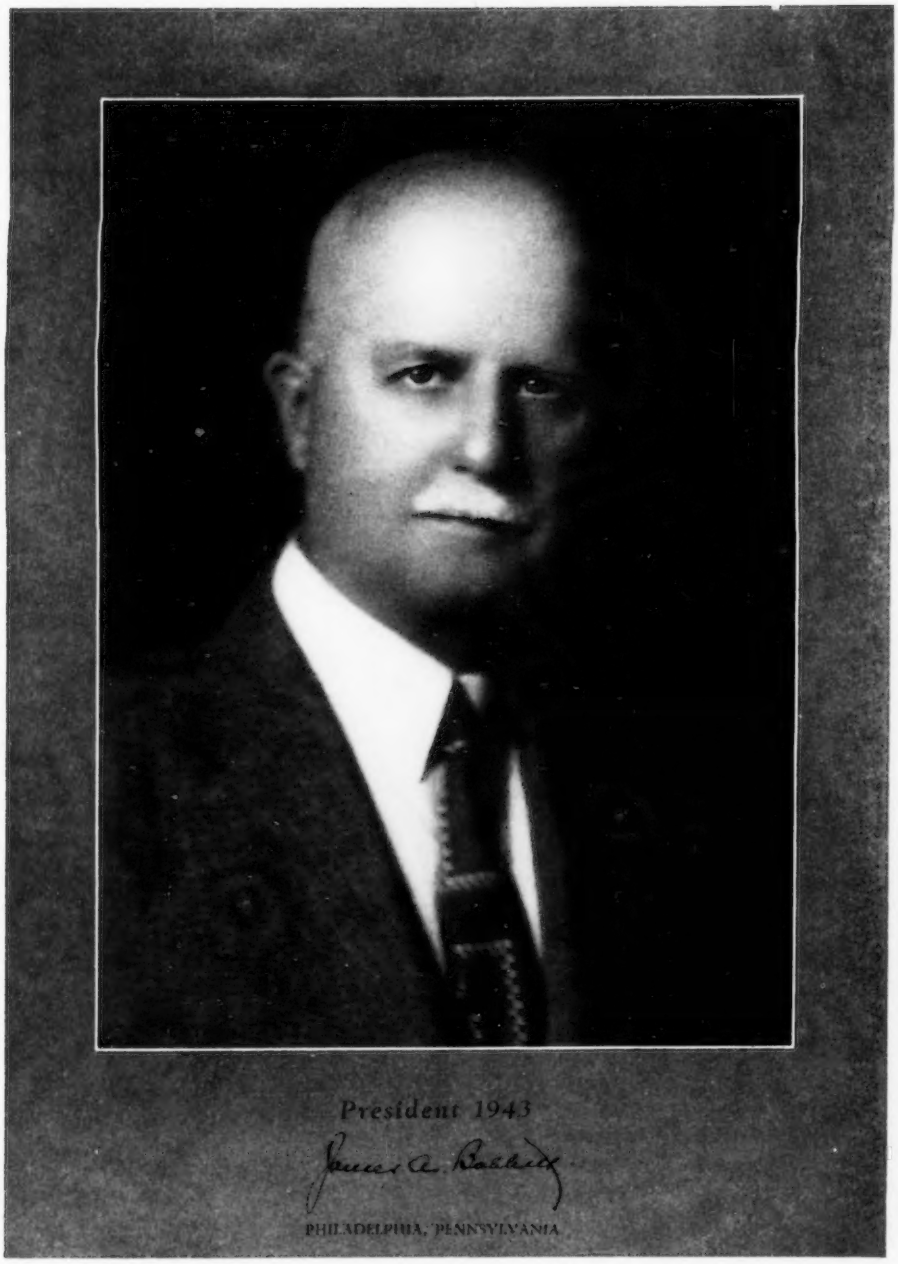

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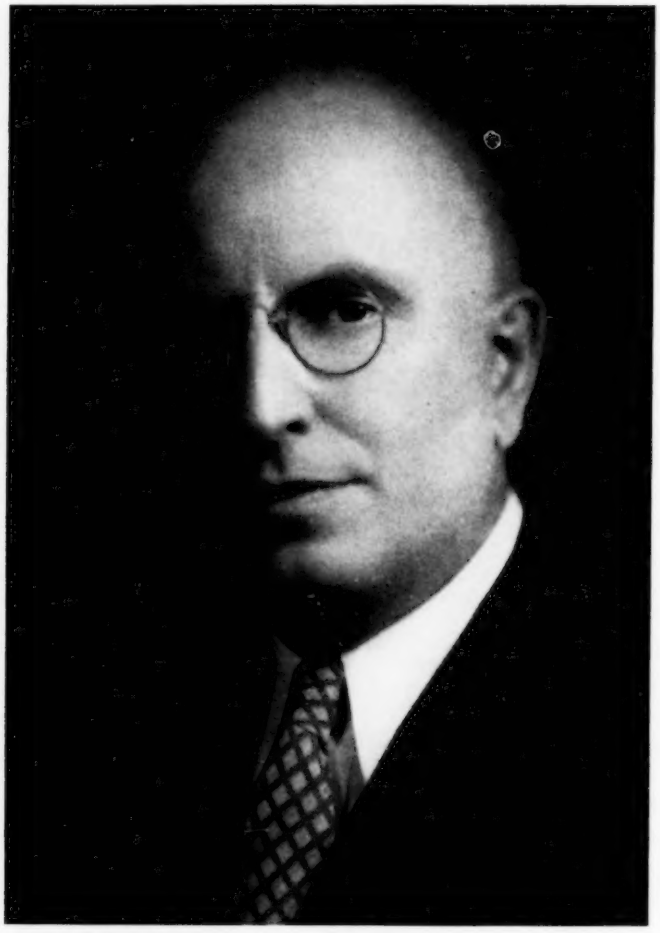

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Address of President

* * *

OUR ACADEMY

JAMES A. BABBITT, M.D.
PHILADELPHIA, PENNSYLVANIA

GENTLEMEN of the Council, Honored Guests and Fellow members of the Academy:

During its life span of nearly half a century, our Academy has weathered many a critical period — international war, financial depression, political and economic upheaval — yet no internal friction of any consequence would seem to have disturbed the almost unique cooperation of its two so distinct specialties, with alternate allocation of the presiding office. It has rapidly developed into a great forum of exchange in ophthalmic and otolaryngologic ideas, fostered by the refreshing intimacy of younger and older men, so utopian a Fellowship that they should be termed Fellows instead of members of this great organization.

Now the global war has precipitated a supreme national crisis, involving many a difficult readjustment. This meeting and its program represent the culmination of a most careful year of planning, with the major obligation that of service to the armed forces. We have indeed been fortunate in that the mantle of the never-to-be-forgotten Secretary Wherry has fallen upon the shoulders of so able an administrator, Doctor Benedict.

The responsibility of an opening address weighs heavily upon a presiding officer from the moment of his induction, and he becomes acutely conscious of his obligation to present a stimulating and constructive message. He realizes how much of the Academy success has been due to the vitalizing heritage from its previous executive leaders.

During the past two decades, ten opening addresses seem to have been devoted to a general analysis and discussion of Academy problems, four to historical reviews and six to scientific presentation. It is obvious that the latter should be based upon active scientific research and not be a mere collaboration of opinions. The majority of these addresses carried a message concerning the Academy, and this one should conform to the pattern.

It may be presumptuous for even a retiring president to attempt a critical review of Academy history, analyze its present or forecast its future, yet it may be appropriate to draw from the background of each enough to interpret its place today, and from an intimate view of the players upon its stage, note important trends and opportunities.

Be assured that the forthcoming suggestions are from one so wholly loyal to Academy traditions that he can visualize but two great groups of arrived ophthalmologists and otolaryngologists in America — those *within* and those *without* the Academy fold, but understand that its doors are open to all candidates of merit. With your permission, this introduction will be followed

Presented at the Forty-Eighth Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 10-13, 1943, Chicago, Illinois.

Department of Otolaryngology, University of Pennsylvania Graduate School, Philadelphia, Pa.

by a brief discussion of three important phases of Academy life—*Guardianship, Education, and Service.*

1. *Guardianship* — Perhaps the exact formula by which the Academy thrives varies much with individual opinion and is contingent upon each member's response to Academy spirit and service. Basic, of course, is the guardianship of its honor, integrity and traditions—the responsibility of each newly elected Council — a group of men tried and loyal in long continued service. Its administration set-up has required little change in recent years. Basic also in its guardianship must be the prevention of any imbalance in the present delightful coordination of the ophthalmic and otolaryngologic divisions of its larger group. Would it be permissible to review these unwritten laws or factors in its smoothly running machinery?

The Academy is conservative but nevertheless carefully and constantly progressive. Majority and not individual opinion rules. It never permits entangling associations to modify its freedom of action. It assumes no dangerous responsibility for individuals or groups within or without its official family. It will harbor no subversive influence where such can be recognized. Its careful financial management, inaugurated by Comptroller Large, has rendered it free from any influence of controlling charity. It accepts no candidate in membership who has not become a diplomate of the Ophthalmological or Otolaryngological Board, with their searching and efficient examination — indeed these Boards are the children of the Academy. The brilliant group of teachers within its ranks are generous in their voluntary courses of instruction.

To your speaker, it rings true as neither political nor selfish. Does not this description in a way pattern the tradition we seek to guard?

2. *Education* — Education per se has too often proven a cloak for intellectual inertia, but not so in the Academy. Your speaker's first and lasting impression was conveyed in the privilege of obtaining a half dozen cards, at trifling fee, to instructional courses twice each morning, now increased to three, at the annual meeting. These cards, thanks to careful preliminary pruning by the Academy management, proved an open sesame to brilliantly concentrated and informative courses by our leading Academy specialists.

It has always been an inspiration to see here and there the matured and respected chiefs of medical departments throughout the land, squeezed in elbow to elbow with the younger specialists, in the crowded class rooms of the corridors allotted to the instructional courses. Their presence is an inspiration to the leaders, to have the courses represent their highest effort. The rush in the halls, in the exchange period between the designated hours, is reminiscent of the excitement of a graduation day in high school rather than a routine group of lectures. To date these courses seem to have lost none of their pristine zest, and the secretaries of instruction merit the highest praise for their careful management.

The success of the single instruction periods has led to more extended, almost laboratory courses, with their microscopic array. From these have evolved the home teaching courses, refreshers for the isolated practitioner, under the enthusiastic, almost missionary leadership of Doctors Gradle, Novak and their associates. The educational scheme of the Academy has included the carefully prepared scientific sessions and choice movie film presentations, and, dignifying all, scientific exhibits which are worth while and amply merit the recognition accorded them.

Probably the question might be raised as to whether or not this finely

correlated educational effort and its munificent offering of refresher courses would stimulate careless individual authority without the adequate apprenticeship and discipline of institutional instruction. This might remove the enthusiasm for graduate courses which our Alumni night gatherings commemorate. The question may be open to argument, but it should be borne in mind that our yearly increment of new membership has passed the respective Board examinations, a prerequisite for which is the preliminary graduate training or its equivalent. This Academy instruction has proved a genuine stimulation and would be by many accorded its major asset.

On the other hand, the Academy has had a vital influence in the establishment of graduate courses and schools throughout the country and has influenced much the proper alignment of undergraduate teaching in the basic fundamentals of our specialties.

These war years are sure to have a profound influence on education in our colleges and medical schools. Medicine itself appears somewhat transitional. This unified Academy forum may be of a profound assistance in the molding of medical opinion and may yet carry out a teaching function in conservative and constructive protection to its specialized department of American medicine.

Another important matter demands thoughtful consideration. This Academy has always sponsored genuine research. The array of standing ophthalmic and otolaryngologic committees in the Council organization attest its research spirit in the conservation of sight and hearing as well as rehabilitation in their defects. Perhaps the time is now ripe for more basic research, even supplanting some of the multiple instruction courses.

In response to query as to suggestions of importance for this meeting, Doctor Mosher, whose presence honors our annual meeting, expressed his concern for some finality in the evaluation of the medical miracles ascribed to blood plasma, chemotherapy, vitamins and aural fenestration, which only time, research and adequate statistics will reveal, and he permitted me to quote him. Most of us will be in complete harmony with this concern. Probably the progress and understanding derived from the experience of the armed forces in the management of the sulfa drugs, penicillin, gramicidin and their derivatives, in the fight against infection, will aid the solution of these problems.

A step inside one of the great base hospitals is most illuminating. Picture the 2,000 or more rapidly changing patients, special treatment of every sort handled by able specialists with subordinates and assistants perfectly organized and disciplined. This group is not pressed by the time element, is not disturbed by volitional confusion as in so many of our hospitals today—here orders are given and obeyed. Equipment is unlimited and of the best, with records complete, not hasty, and with abundant time for adequate clinical conference. This means that war medicine is going to take prodigious leaps in the tremendous lessons of mass experience. Its officers are proud of their work and do not need civilian help at this stage. However, the returning Medical Corps will doubtless be only seasoned in certain types of ear, nose and throat surgery and treatment. There will be a wide margin of service left.

A gigantic field of research is still open, and opportunities for meticulous collaboration of real statistical value, both favorable and unfavorable, are equally urgent in their invitation.

Is it possibly the time now, and if not, in the post-war regeneration, for our Academy to concentrate more of its time and resources upon these fields?

3. *Service* — Service in the terms of this message means Service to the Armed Forces, Service to the Academy, Service to the country at large, Service to our individual members.

1. We hope you realize how earnestly the Program Committee has endeavored to focus this meeting on the Service to the Armed Forces. The symposium on Youth Development in America, post-war rehabilitation from impairment of sight and hearing, plans for stimulating the Inter-American good neighbor relationship, specially pertinent instruction courses and moving picture demonstrations all converge upon this purpose.

2. Probably Service to the country at large is mainly through educational influence and published propaganda. It is likely that through this means, our organization contributes much to America and American medicine.

3. Service to the Academy has already been conveyed in the appeal for Guardianship. The Academy is what it is from the continued loyalty of its members. This support is often intangible and obscured but very real, and we bespeak its continued expression.

4. Under the title of Service to our fellow ophthalmologists and otolaryngologists, it would be appropriate to introduce a topic of major importance today — the future field of activity in these two great groups. This may not concern both, but it is vital to one, and lest he tread on unfamiliar ground, your President's parting message will be directed to otolaryngology.

A thoughtful graduate student, after a recent lecture, expressed his concern in this fashion. Biochemistry (referring to sulfa and other anti-infection drugs) bids fair to absorb the therapeutic control in the pharyngeal, accessory sinus, middle ear and mastoid fields "sans" surgery. The allergic and bronchoesophagoscopic specialties will supplement the list. General medicine will challenge the fringe and push "spearheads" into all phases. What is there for me to look forward to as a specialist?

This man obviously did not want to conduct a "nasal laundry," to quote an apt term suggested by one of our Academy leaders, or to cultivate addiction to useless tubal ear treatment, and he probably was uncertain in his surgical field. A reply outlining the great opportunity of research in impaired hearing, including fenestration and neuro-otologic surgery, scarcely appeased him. This episode inspired the title of a talk before a local society in Pennsylvania, by your speaker, on an Otolaryngologic Forecast, with suggestion of detours necessary to avoid this "dead end" or "bottle neck" in competition with medicine and surgery.

Curiously, in Doctor Mosher's communication, referred to above, he came to the same conclusion and expressed it with the suggestion that "one be bigger than his specialty."

The great answer and prophecy for a successful detour for you established specialists is to burst the complacency of rather sordid routine. Freshen up the medical background of everything concerning both upper and lower respiratory tract, restudy Materia Medica, therapeutics, anatomy, physiology and pathology. With this equipment, become what the world needs today, more and more finished all round surgeons, competent to perform all surgery of the head and neck which should rightfully be done. Why should not this include thyroid, deep neck, meningeal and major plastic surgery?

Many of us need to review our first principles in surgery — topographical anatomy, convincing ability in the confident skin incision, revamped ability in deft plastic suturing, perfect aseptic and antiseptic technic, control of a bloodless field, acquaintance with the name as well as use of every instrument. Rhino-

plasty, facionaxillary surgery, neuro-otology, deep neck surgery, bronchoesophagoscopy and tracheotomy, with allergic and Barany reaction mastery, are yours for the asking. Academy service can aid in its accomplishment.

Your speaker is convinced that the specialist in our field must become a better medical man and a greater surgeon. Accept the prophecy that this *will* happen, as a final forecast.

In closing, may I express my heartfelt appreciation for the honor you have accorded me, for the generous support of the Executive Officers of the Academy and the Chicago local committee.

Address of the Guest of Honor

THE UNITY OF MEDICAL SCIENCE IN THE AMERICAS

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PORTLAND, OREGON

HERE is a pretentious title for a few observations respecting the historical backgrounds of our common interest in medical education and in our hopes and aspirations for future medical progress. Perhaps it may be salutary even if a bit unpalatable to look at ourselves and at our own medical development through the eyes of our neighbors; to find out what their histories say about us, and something of the reasons for our differences in cultural development and outlook.

America, South and North, forms a great island, which Columbus thought to be Asia. We are protected, as is no other continental group, by sea and distance, from outside aggression. The history of the nations of South and Central America and Mexico, involving our own southern borders and the Pacific Coast as far as Alaska, was not merely the story of armed exploiters seeking gold and silver; it was, rather, the transplantation of contemporary Spanish and Portuguese culture to a new world. Devoted missionary teachers, who were the scientists and medical men of that distant day, founded schools, hospitals and universities, printed books, compiled and translated native languages, and tested and catalogued native medicines and plants. These good men endeavored to protect the natives from enslavement and exploitation by the conquerors, and ministered to them when infectious diseases from Europe became epidemic.

The Spanish renaissance was stimulated by the reconquest of the Iberian peninsula from the Moors and the union of various small kingdoms under Ferdinand and Isabella in the middle of the fifteenth century. Gleaning the best of the brilliant Moorish civilization, particularly in medicine and other branches of science, Spanish scholars and their universities led Europe for a hundred years. In the words of Ramón y Cajal, whose historical judgment paralleled his skill as a neuroanatomist, "the highest peak of achievement for Spain in her own interest was reached in the glorious reign of the Catholic monarchs." The powerful influence of this culture is reflected in the solid churches, hospitals and other public buildings still in use in every Latin-American country.

Eight universities were founded in Latin America before Harvard came into being in 1636. St. Thomas Aquinas (1538) in Santo Domingo, no longer exists; but St. Mark, Lima, Peru (1551); México (1553); Córdoba, Argentina (1614); and Sucre, Bolivia (1623), are still at work. The first school in Mexico was established in 1553, the first printing press in 1535. The first medical work published in the new world was the Opera Medicinalia of Francisco Bravo, published in Mexico City in 1570, and was followed by works on surgery and medicine by Alonzo Lopez and A. Fárfan, between 1578 and

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1592. The binding and typography of these and subsequent works was comparable to the best work from Spain of the same period.

North American colonization a century later arose from the desires of limited numbers of relatively poor and humble groups to establish themselves in new regions free from religious strife and class domination, as well as from the establishment of trading companies chartered by royalty. Colonization of Latin America was radically different, involving the actual extension of the governments of Spain and Portugal to the rich new land. Because of dissension among the discoverers and settlers, on the African and East Indian coasts as well as in America, Pope Alexander VI in 1494, established a line of demarcation which limited the Portuguese to Brazil and Africa; the Spanish had all the rest of America. The Portuguese were scattered on a lengthy tropical coastline, and worked up the Amazon as far south as the River Plata. Though subjected to intermittent attacks by the Dutch, French, Spanish and English, their ties with the mother country remained so strong that the royal family fled to Brazil during the Napoleonic wars; and out of this grew the Brazilian empire, which flourished during the rule of Don Pedro II, himself a scientist of repute.

The Spanish explored North America as far north as Kansas, and up the Pacific Coast to the west shore of Vancouver Island, and their influence remains in Florida, Louisiana, Texas, New Mexico and California. These vast areas were ruled by viceroys from Mexico, while the viceroys of Peru controlled all of South America but Brazil.

Early insistence upon the rights of native peoples was broken down under later colonial governors, as the power of Spain declined with the rise of France and England in the seventeenth and eighteenth centuries. Nevertheless actual slavery by importation from Africa was not permitted. Many natives secured excellent training through entrance into the various monastic orders which controlled education. Such men of native ancestry were active in the eventual revolt against corruption, exploitation and cruelty by the last degenerate representatives of European control. Two such native priests were early heroes of the war for Mexican independence.

Just as charlatans of various sorts preyed on the frontier settlers of the Western Reserve, Tennessee, Illinois and Missouri in the early days of the nineteenth century, similar quacks were found in the early Spanish and Portuguese outposts. A few licentiates from Spain, and other doctors from Portugal, Switzerland and Holland began to establish themselves in practice after 1610. The Jesuits, skilled in medicine, were driven from Brazil in 1599 for political reasons and founded a remarkable cooperative colony in Paraguay. Their missions, similar to those of California and the southwest, were later turned over to other monastic groups less skilled in medicine. Their seminary in Córdoba became a university in 1614, but political complications in Europe caused their expulsion in 1767.

The first department of health was established in Buenos Aires in 1780 by Dr. Miguel Gorman, of Irish descent, who had been trained in Paris, Rheims and Madrid. A man of strong character, he fought the charlatans and introduced vaccination. He instituted examinations for qualification to practice, and began a six year medical course in 1801. This pioneer work was interrupted by the defensive campaign against British invasion in 1807, as well as by the jealousy of other local physicians. Doctor Gorman died, blind and infirm, in 1816. Finally Rivadavia, the national hero of Argentina, re-established the university and medical school in 1821.

Another pioneer figure, Hipólito Unanue, was an eminent physician, medical teacher and patriot, in Peru. He reorganized medical teaching at Lima, following the model of the school of Leiden in Holland, and founded the Colegio de Medicina de San Fernando. He died in 1833.

In every country to the south such educated men as these took counsel together when the American revolution was successful. They became leaders in similar movements for freedom from European domination. Representatives of Brazilian patriots and of other South American groups, eager to get away from foreign control, asked Thomas Jefferson, then our minister at Paris, for advice and for help from the United States. This he was unable to give them, but his letters betray his sympathy with their ambition to be free. They feared both France and England; but after the Napoleonic collapse, distrusting England even more, they turned from the United States because our colleges were too new, and because we would not learn their language. Our war with Mexico, which they stigmatized as an attempt to extend slavery to Texas and the southwest, alienated them still further.

We should perhaps be reminded that America to the south of us is composed of numerous soverign nations, between which rivalry exists and occasional conflicts have occurred. These nations are naturally proud of their indivdual independence, their history and their heroes. Similarly, their medical institutions are supported with deeply nationalistic pride, and their medical societies and periodicals are national in scope. International congresses have been hard to arrange, not only because of the considerable distances between medical centers, but also to some extent because of the difference in language between the Brazilian profession and that of the other countries. Remembering our fidelity to our own medical schools and graduate institutions, we should look with a good deal of sympathy upon the pride of our northern and southern neighbors in their own admirable schools, hospitals and professors.

Medical schools in Latin America followed continental European models, and their advanced students went to Paris, Rome, Bordeaux, Leiden; rarely to Barcelona or Madrid, never to London. Rebuffed by our tendency to magnify our own achievements, sensitive over our so-called "dollar diplomacy" and our failure to learn their languages or cite their published work, our colleagues to the south turned increasingly to European clinics for study in the last twenty-five years. Graduate teachers in France, Spain and the Germanic countries were not slow to take advantage of this situation; many settled in South and Central America, a few in Mexico, and certain eminent teachers went there repeatedly to lecture.

One should not forget that a similar language problem and cultural conflict has long existed in Canada. The French-speaking profession of Quebec has maintained its own medical schools and hospitals, and does not participate in the Canadian Medical Association. Many French Canadian physicians went formerly to France or Belgium for graduate study; now, not a few are to be found in the northeastern United States. With new bonds of fellowship arising from the present conflict, continuing friendliness between the two language groups in the Canadian profession seems probable.

What shall be the attitude of the professon of the United States toward our medical neighbors? We have for years sought to help the physicians and health institutions of Latin America. The trustees of the American Medical Association published a Spanish edition of the Journal at a heavy loss for several

years; this project has been revived recently and awaits the cooperation of the Department of State. This Academy, the American Medical Association and the American College of Surgeons have for years past invited leading practitioners from the north and south of us to present papers on our programs, and we have benefited from valuable contributions coming from splendid schools and institutions.

Our colleagues to the south have deeply resented attempts by Spanish, Nazi and Italian groups to influence or control them, or to secure expressions of allegiance, cultural or quasi-political, to the so-called mother country of Spain. They have not been taken in by the present Fascist government of a country from which all of them had secured their independence more than a century ago. They have the pride of more than four hundred years during which their peoples have developed civilizations independent of Iberian rule; and to the original native stock and the Spanish and Portuguese settlers have been added great numbers of other Europeans, particularly Italians and Germans in the more temperate zones of the south. Geographically Americans like ourselves, maintaining their own individual racial melting-pots, our neighbors very properly resent any attitude of condescension or self-aggrandizement on our part. They are sincerely grateful (as in Ecuador, Brazil and Mexico) for the fine effort which the Rockefeller Foundation, the Army Medical Corps and the Public Health Service are making to help them with their problems of transmissible and epidemic disease, sanitation and water supply.

We have a tendency to exaggerate our own achievements, and, being human, at times to depreciate the work of others. We should be more familiar with the splendid work done in Latin-American schools. It should not be forgotten that Carlos Finlay of Havana outlined the probable cause and control of yellow fever in 1881, many years before Walter Reed and his associates risked their lives to prove it.

The language barrier must be overcome, whether by exchange of students, by exchange lecturers or otherwise; certainly by familiarizing ourselves with the excellent work which is done outside our borders and reported in the Spanish and Portuguese journals. Proposals are already being made in South America to form examining boards in the specialties, patterned on our own, by which so many of our Canadian colleagues have already been certificated.

On our two free continents there is no interference by government with the free choice of one's physician; no totalitarian attempt to change the curriculum of medical instruction; no government-controlled medical institutions staffed by incompetent political appointees; no transfer of medical personnel from city to city by government decree. We must deserve this freedom, planning for the future health and welfare of all our citizens; remembering, in the emergency of war, that we must not neglect the responsibilities of peace. Thousands of our colleagues are with the armed forces; we must not stand aside from public duties in which we must represent them, such as participation in state medical societies and boards of health, and particularly in state legislatures and in the Federal congress. Many physicians among our southern neighbors have, as educated leaders, served their nations as lawgivers, and a number have attained presidential office. Perhaps we should not look so high. When, however, untrained, self-seeking and uninformed groups seek to gain control of the management of the health of our citizens, it is time for people of practical experience in care of the sick to take definite action. There is

illustrious precedent for the physician to enter public life; recall that Benjamin Rush, renowned Philadelphia doctor, teacher and epidemiologist, was active in founding this republic, and was a signer of the Declaration of Independence.

Certainly we should be first to admit that we have no monopoly on wisdom. If we are to step through the looking-glass and examine ourselves without prejudice, and with due regard to the high achievements of our brethren to the south and to the north, we shall, perhaps, eventually deserve what President Thomas Jefferson wrote in 1807 to Dr. Caspar Wistar of Philadelphia:

"The anatomical and clinical schools are those in which the young physician should be formed . . . . I consider them as the two fundamental pillars of the edifice. . . . It is from this side of the Atlantic, that Europe, which has taught us so many things, will at length be led into sound principles in this branch of science, the most important of all others, being that to which we commit the care of health and life."

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Address of the First Vice-President

* * *

AN EVALUATION OF OCULAR ANGIOSPASM

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PENDULUMS, as is well known, have a way of swinging. When an idea has served to explain one condition satisfactorily the attempt is invariably made to use it for other purposes and the pendulum is set in motion. It behooves anyone who has given any pendulum even a mild push to go back, now and then, and see whether it is resting in a physiologic position, or in one which defies the laws of gravity.

The conception of angiospasm is by no means new but the attempt to explain a number of pathological ocular conditions as due to that entity is largely a work of recent years. My own interest in the subject began with a study of Krogh's[5] work on the capillaries, published in 1922, the Atlas of capillary microscopy by Müller,[10] which appeared in 1922, and some applications of this work to ocular conditions by Scheerer, Parrisius and their associates[12] in Tubingen. Out of this work arose the conception of a *vasoneurotic diathesis.* When the peripheral circulation was studied by various methods, certain individuals were found to have an abnormally labile vasomotor system. In response to various stimuli, especially cold, certain drugs such as nicotine, and in some cases certain foreign proteins, these individuals showed signs of spasm involving the peripheral vessels with resulting stasis in the capillary circulation. The capillaries themselves, although devoid of a true muscular coat, are narrowed, as Krogh has shown, by contraction of the Rouget cells in their walls, and in cases of the vasoneurotic diathesis such contractions are easily provoked and are abnormally marked and prolonged.

In the earlier work reliance was placed chiefly on capillary microscopy and patients with various ocular conditions were studied by this method. It was thought at first that patients with glaucoma often showed signs of the vasoneurotic diathesis but the evidence was by no means convincing and Mielke[9] found no such relationship in his series. Attempts were made by Scheerer and others to determine vasoneurotic signs by a study of entoptic phenomena but these efforts were not particularly successful.

As the study of peripheral vascular diseases has progressed, there have been developed a series of observations and tests which will determine with a fair degree of accuracy which patients have a pathological degree of peripheral angiospasm. I am deeply indebted to Dr. Gilbert Marquardt for information concerning these special methods of examination and to him, as well as to Drs. Geza de Takats, Theodore Van Dellen and J. F. Mallach, for data on the patients sent to them for examination. Few ophthalmologists are qualified to make these examinations and it is unfor-

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tunately true that many internists are not sufficiently interested in the subject to provide the desired information. The examination should include a careful history. Vasoneurotic individuals have usually noted undue distress on exposure of the extremities to cold. Their hands become white when elevated and flushed or even cyanotic when dependent. They take particular care to protect the extremities against cold so as to avoid various unpleasant symptoms.

Examination should really be carried out in an air-conditioned room but it is usually sufficient if the room is free from drafts and if the patient is allowed to rest in such a room for a half hour before examination. This is to prevent the rapid changes in findings which occur in vasoneurotic individuals when exposed to temperature changes. Observation of the extremities confirms the statements in the history. The hands and feet are pale, and readings with a skin thermometer show an abnormal difference between the oral temperature and that of the extremities. A difference of three to four degrees Fahrenheit is normal, while vasoneurotic persons show differences as high as 10 degrees. The use of nicotine, as in smoking a cigarette, will cause a further drop in the peripheral temperature of susceptible individuals of six to 12 degrees. Normal persons may show a drop of four to five degrees under these conditions. In one of our patients this drop amounted to 13 degrees Fahrenheit. Oscillometric readings are low. The pulse in one or both posterior tibial arteries may be weak or imperceptible. Capillary microscopy of the nail-folds shows a narrowing of the arterial limb of the capillary loops with stasis, small aneuryms and hemorrhages in a number of areas.

When the above tests have been completed and the temperature of the extremity has returned to its original figure, blocking of the posterior tibial nerve is employed in patients showing signs of spasm. In patients without organic changes in the vessels, which includes most of the younger patients in this group, the temperature rises following nerve-block so as to approximate the oral temperature. In cases with arteriosclerotic or thrombophlebitic changes the rise in temperature is less or may be absent.

The blood pressure of the younger individual in this group is low or normal. Following the cold pressor test (dipping the hand in ice water) little or no rise in blood pressure occurs. When a rise of pressure does occur, the case is considered to be one of early or latent hypertension and hence in a different class from the vasoneurotic individuals in whom we have been most interested. The factor of peripheral spasm in patients with hypertension is of great importance, but I wish to emphasize here the fact that a number of young persons show marked peripheral angiospasm without elevation of the systemic blood pressure.

A few other tests may produce information of value. Blood cevitamic acid often reveals low figures even in persons on a normal diet. Basal metabolism is low and blood cholesterol high in a few cases, and the amount of noniodizable calcium in the blood may be low. There may be a relative achlorhydria, according to Marquardt, although gastric analysis was not done in most of our cases.

Undoubtedly there are a number of vasoneurotic individuals who show no ocular pathology and in certain patients showing ocular pathology of various types, a tendency to peripheral angiospasm may be revealed which has no relation to the ocular condition. My reference to the swinging of the pendulum was prompted chiefly by the work of Duggan.[2] He has emphasized the factor of angiospasm in a variety of conditions, many of which have been considered, for what seemed good and sufficient

reasons, to be of inflammatory origin. While local vascular spasm may play a part in the edema which accompanies inflammation, Duggan's work seems to have resulted in a certain confusion. Attention has been directed away from a search for the causative factor in these conditions, and the supposed effect of treatment by vasodilators has, it seems to me, been unduly emphasized. Most of his cases were not submitted to the pressor tests described above, but were treated empirically.

It is the purpose of this communication to describe a few fairly well defined types of ocular pathology, chiefly two, in which peripheral angiospasm seems to be the causative factor, and to urge that patients presenting signs suggestive of these conditions be submitted to a type of examination which will determine whether or not abnormal peripheral angiospasm is present.

The most important of these conditions is what Marquardt and I[3] have called Central Angiospastic Retinopathy. In this we have followed Horniker, but have preferred the term retinopathy to retinitis. The term retinitis seems to us especially unfortunate since it is our conviction that the condition is definitely not inflammatory but due entirely to a circulatory disturbance.

In our previous communication the literature was reviewed, and reason even given for identifying this condition with that described by a number of previous authors under various names. These included central recurrent retinitis (von Graefe), retinitis centralis (Asayama), retinitis centralis annularis (Kraupa), choroiditis centralis serosa (Riehm), pre-retinal edema (Guist), idiopathic flat detachment of the retina (Walsh and Sloan). In most of these previous reports, the condition was thought to be inflammatory and, as usual, tuberculosis was invoked as the probable cause. This was done by the reporters of a remarkable series of cases in Japan. Matsuoka, for example, collected reports of 640 cases, and Kitahara reported 150 personal cases. Knowing what we now do of the condition, we may add to various more or less scientifically verified impressions of the Japanese the conviction that they certainly must be a spastic little people. A few authors such as Kraupa and Fuchs suspected the circulatory origin of the condition, but it remained for Horniker[4] in 1927 to 1937 to look for other signs of angiospasm and to find them in most of his 42 cases. His methods of examination were not so accurate as those at present employed, but the response of his patients to the use of sedatives and vasodilators must be considered additional evidence that the condition he described was due to spasm of the smaller retinal vessels or, in some cases, of the capillaries themselves.

Our report dealt with eight cases. Six of these were typical in that only the area of the macula was involved, while in two some of the larger retinal vessels were also occluded. Special examination of the peripheral vascular system was made in only six cases and all showed evidence of angiospasm (Table I). All eight were men between 17 and 43 years of age. Nearly all smoked. The typical picture was sudden reduction of central vision. This varied from a slight loss with small relative central or paracentral scotoma to a reduction to 20/200. Either one or both eyes were involved and often there was a history of previous attacks. Metamorphopsia was a common symptom in cases with slight visual loss. During the acute attack the predominant fundus change was edema involving either the whole macular area or some part of it. (Fig. 1.) With the fine beam of the Friedenwald ophthalmoscope this area was seen to be distinctly elevated, as shown by a bending of the beam. The splitting of the beam described by Walsh and Sloan which led them to diagnose their cases as flat detachment of the retina was not seen, and for a number of

reasons it was considered that swelling was present in the retina itself, particularly in the nerve fiber layer. The transient hyperopia observed in several cases would suggest an actual elevation of the retina in these cases. A striking feature in several cases was a pronounced circular reflex surrounding the macular area, and localization of this reflex with the fine beam seemed to confirm Guist's opinion that it represented an elevation of the internal limiting membrane by edema fluid. Small hemorrhages were seen at the ends of the visible vessels in only a few cases. The larger retinal vessels were normal in most cases, although a few showed constriction of one or more vessels, and in one case obliteration of several branch arterioles had occurred. The occurrence of the edema without involvement of the visible vessels was what led to the conclusion that the capillaries themselves were involved, an opinion shared by Horniker and Bailliart.[1]

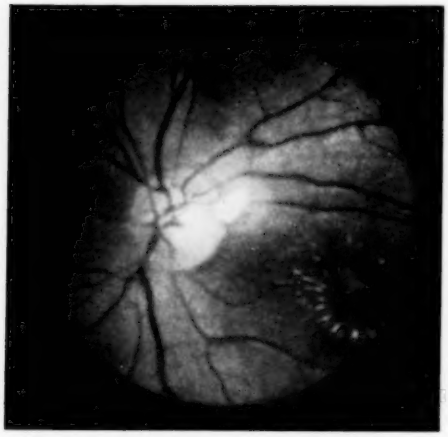

FIG. 1 — Central Angiospastic Retinopathy — acute stage.

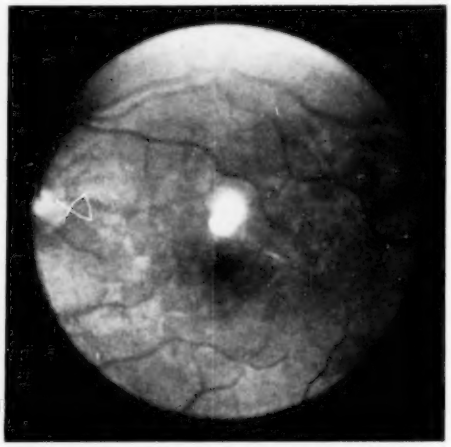

FIG. 2 — Central Angiospastic Retinopathy — healed stage.

In all of the first eight patients, except one, restoration of normal vision occurred within two to six weeks in response to what we called antispasmodic therapy. The edema was replaced in these cases by a very fine absorption of retinal pigment which sometimes involved an area larger than the macula. (Fig. 2.) In some cases fine granular pigment appeared in the macular area; in one case the choroidal vessels became visible, with a line of pigment about the defective area. In a few cases, in which subsequent reports were furnished by other ophthalmologists, the fundi were said to have returned to normal, but in all cases followed personally, signs of absorption of the retinal pigment were observed. In a number of cases, the fine white spots, seen by Horniker and Bailliart and erroneously described by them as Gunn's dots, were seen.

In none of these eight cases have recurrences been reported. Most of the patients continued to refrain from smoking and some continued on moderate antispasmodic therapy for a considerable time. On this therapy most patients gained weight and reported improvement in general health.

Since this early series, I have seen 23 additional cases which it was felt belonged in this category. Fifteen of these were sent to Drs. Marquardt, Van Dellen, Mallach or de Takats for pressor

TABLE I

Results of Special Examinations in the Reported Cases

Case No.	Blood Pressure, Average	Oscillometric Data on Ankle: Before Treatment	Oscillometric Data on Ankle: After Treatment	Temp. of Toes Before Nerve Block	Temp. of Toes After Nerve Block	Degree of Capillary Spasm	Acrocyanosis of Toes	Reaction After One Cigarette	Treatment
2.	118/80	3.0+	3.0+	32 C. (89.6 F.)	34 C. (93.5 F.)	++++	—	Drop of 4.6 degrees C. (8.2 degrees F.)	Stopped smoking; phenobarbital.
4.	124/80	2.0	3.0+	28.6 C. (83.4 F.)	32.4 C. (90.3 F.)	+	+	Drop of 4 degrees C. (7.2 degrees F.)	Tissue extract; phenobarbital; thyroid extract; Stopped use of tobacco.
5.	130/80	1.5	—	27.4 C. (81.3 F.)	33.2 C. (91.7 F.)	++++	++	Drop of 5 degrees C. (9 degrees F.)	Stopped smoking; phenobarbital.
6.	120/70	2.0	2.0	33 C. (91.4 F.)	35.2 C. (95.3 F.)	++	+	Drop of 3.2 degrees C. (6.7 degrees F.)	Stopped smoking; phenobarbital; typhoid vaccine intravenously; tissue extract.
7.	144/90-104/68 Low reading after nitrite therapy	1.5	3.0+	25 C. (77 F.)	31.2 C. (88.1 F.)	++++	++	Formerly smoked; drop not measured	Nitrites; tissue extract; typhoid vaccine intravenously; treated 6 days, stopped smoking.
8.	128/82	1.0+	3.0+	29.2 C. (84.5 F.)	32.8 C. (91 F.)	++++	++++	Drop of 4.6 degrees C. 5 minutes	Tissue extract; phenobarbital for 6 months; stopped smoking.

tests. Sixteen were seen during an acute attack with the typical picture just described. Five were seen only after the edema had subsided and showed the slight degenerative changes described in this post-spastic period. Of these 23 cases, all which were followed showed recovery of normal or nearly normal vision. In a few cases vision remained reduced to 20/30 or 20/25 with persistence of a small relative central scotoma. Several patients were seen only once in consultation and were not followed. In two cases the changes were more severe, and such as to cast some doubt on the diagnosis, or to modify the picture of the condition previously formed. One of these patients, a woman of 31, presented a typical fundus picture when first seen, with the visible vessels normal and an extreme macular edema with radiating white deposits approaching in appearance a macular star. The blood pressure was normal. Vision was reduced to 5/200 and after three months had only returned to 20/100. Some macular edema was still present.

The second was a woman of 30, observed by Dr. Roy Riser for more than a year. The sharply circumscribed macular edema with circular reflex was typical, but fundus pictures taken during this time showed practically no change in the condition. Vision could be improved by a + 3.00 sphere to 20/25, though a relative scotoma was present. The other eye was normal. Pressor tests showed little evidence of angiospasm.

The ages of these 23 patients, with one exception, ranged from 25 to 45, averaging 36 years. The exception was a man of 61, with normal blood pressure, marked pressor reactions, especially to tobacco, and recovery on antispasmodic treatment with abstinence from tobacco. Whereas in the early series only men were affected, of these 23 cases eight were in women. A number of these women smoked, and a few to excess. Their ages ranged from 28 to 40. One showed, in addition to the signs of peripheral angiospasm, signs of pseudo-Addison's disease with abnormally low systemic blood pressure and minus 20 basal metabolism. In addition to these 23 cases, there were at least four cases in which spasm of the fine vessels supplying the macula appeared in association with generalized arterial hypertension. Other signs of peripheral angiospasm were present in two of these, the only ones submitted to the tests, and antispasmodic therapy seemed to be of benefit for the ocular as well as the systemic condition, although restoration of function was never so complete as in the younger patients without systemic hypertension.

Due to the absence of Drs. Marquardt and Van Dellen on military duty, and the fact that examinations were conducted in several offices and hospitals, it is impossible to prepare a table of vascular findings including all the points covered in our first report, and in some cases the data would seem to be incomplete. Of the 15 cases referred for special examination, however, all but two showed some evidence of abnormal peripheral angiospasm. One of these, a male, was seen with a small healed lesion resembling those developing later in this group. He never showed signs of retinal edema during observation. The other was a man of 42 whose pressor tests were negative, but who gave a history of marked discomfort on exposure to cold and showed a basal metabolism of — 22. He did not smoke. His condition cleared completely on thyroid extract and injections of tissue extract. Re-check during antispasmodic therapy in a number of cases showed an improvement in the degree of angiospasm, the findings in some cases approaching normal. It is recognized, however, that the tendency to peripheral angiospasm is, in most cases, a hereditary constitutional anomaly and that

measures to prevent or minimize spasm, especially protection against cold, and abstinence from tobacco, will always be indicated for these susceptible individuals.

The treatment employed in most of these cases, in whole or in part, is as follows:

1. Complete abstinence from tobacco and protection of the body against cold.
2. In the acute stage papaverine ½ grain twice daily by vein for several days, followed by ½ grain by mouth three times a day for one to two weeks.
3. In some cases typhoid by vein was employed for its vasodilating effect. Small doses of 10 to 20 million organisms by vein seemed as effective for this purpose as larger doses.
4. Hypnotics, either combined with a diuretic in the form of theominal one capsule t.i.d. or as phenobarbital ½ grain t.i.d. were continued for periods of several months.
5. Tissue extract. Usually an insulin free pancreatic extract (depropanex), was given by intramuscular injection one c.c. twice a week for 14 injections, the course being repeated in several cases.
6. The nitrites, including 10 per cent sodium nitrite by vein as advised by Duggan, were employed in some cases, but according to Marquardt, the effect of this group on the small vessels or capillaries is neither as marked nor as prolonged as that of papaverine and the barbiturates and especially a combination of the two.
7. The parasympathomimetic drug, prostigmine, was employed in a few cases in doses of 7½ to 15 mg. by mouth t.i.d. and with apparently good effect.
8. Nicotinic acid, either in B complex or in pure form, 150 mg. a day. Its effect on the capillaries is especially marked.
9. A diet containing calcium and the essential vitamins was advised, supplemented where indications were found by concentrates of vitamins C and B complex.
10. In five cases with lowered basal metabolism, thyroid extract was employed, and in a few women showing menstrual disturbance, an estrogenic extract was supplied.
11. A regimen including plenty of rest and avoidance of fatigue and nervous strain was advised where it seemed practical.
12. Checks were made on the pressor tests in a number of cases as an indication of the need for further treatment. Patients were advised against resumption of smoking at any time, and small doses of barbiturates were continued for some time or resumed when signs of nervous tension or insomnia developed.

Only a few reports on this condition have come to my attention since our early report. Duggan[2] seems to have observed six cases of the typical condition described above, with acute attacks responding to vasodilators. He has included as a chronic form of the condition three cases which seem to me doubtful and four cases which he admits fall in the group of disciform degeneration of the macula. This seems to confuse the issue and few ophthalmologists would agree with Duggan's statement that "disciform degeneration differs only in degree from the mild lesions of cho-

roidosis seen in young adults." His preference for the name choroidosis centralis serosa rests upon certain debatable points which need not be discussed here. No pressor tests were recorded in his brief case report, response to the nitrites being evidently considered sufficient to confirm the diagnosis of angiospasm. Schaub[11] reported one case in which the fundus picture seemed typical but recovery was extremely slow. Löwenstein[7] reported three cases and suggested that allergy was a likely factor in certain cases. Smitmans[14] saw nine cases in one year which he diagnosed as central serous choroiditis. All recovered normal vision under treatment with calcium, vitamins and papaverine. The earlier literature was reviewed in our previous report.[3]

Next to central angiospastic retinopathy, the ocular condition, in which the question of retinal angiospasm seems to be of most interest, is periphlebitis retinae with recurring vitreous hemorrhages. Marchesani[8] in 1934 first suggested that patients with periphlebitis, a condition usually considered due to tuberculosis, might have a disease of the retinal vessels comparable to Buerger's disease of the extremities, and this theory seemed to be confirmed by examination of the peripheral circulation in his cases. He reported on 25 patients of whom three had gangrene of the extremities requiring amputation while most showed only signs of "latent" thrombo-angiitis of the extremities as revealed by examination of the extremities and capillary microscopy. Seven of his cases, examined by Stander, showed evidence indicating involvement of the cerebral vessels including oculomotor paralysis. In one of his cases sections of one eye showed thrombo-angiitis obliterans of the retinal and uveal vessels and no evidence of tuberculosis. Closure of retinal vessels has been reported by Lisch[6] and Uyama[15] in cases of Buerger's disease. Lisch,[6] however, on later examining 35 other patients with various forms of peripheral vascular disease including Buerger's and Raynaud's diseases, found none with periphlebitis. Schmidt[13] found no evidence of peripheral angiospasm in eight cases of periphlebitis, all of which showed evidence of tuberculosis.

In view of this somewhat uncertain status of the subject, it seemed of interest to report the findings in a group of similar cases seen since 1939. There have been 12 such cases, of which seven were referred for examination of the peripheral vascular system. No patients with hypertension, nephritis or diabetes were included, nor any in which trauma was a factor. Cases showing evidence of past or present iritis or choroiditis were also excluded.

The cases were picked as showing evidence of severe retinal vascular disease of the type usually called periphlebitis, with, in nearly all instances, spontaneous hemorrhages into the vitreous and a tendency to the formation of pre-retinal bands of connective tissue. Most patients were young adults, the average age being 28.6 at the onset of ocular symptoms. The oldest patient was 42 and the youngest eight. Eight patients were males and four females. In seven cases more or less satisfactory evidence of tuberculosis was found, chiefly positive reactions to high dilutions of tuberculin given intradermally and, in some cases, positive chest findings. These cases will not be further discussed, except for two which showed also evidence of peripheral vascular disease. One patient showed no evidence of tuberculosis and was seen before pressor tests were being advised. Several foci of infection were removed, and he has been free of recurrences for seven years. Five of the seven patients re-

ferred for tests showed evidence of peripheral vascular disease and in three of these it was the only factor found which could be considered of etiologic importance. Two showed no evidence of angiospasm. One was tuberculin positive, while no etiological factor could be discovered in the other case.

The two patients showing evidence both of tuberculosis and peripheral angiospasm may be discussed briefly. One was a woman of 25, with positive tuberculin tests, minus 26 basal metabolism, blood cholesterol 187 mg. per cent, and positive pressor tests, including a drop of 7.2° F. in the extremities after smoking. Her condition cleared up on thyroid and foreign protein injections without any tuberculin, and she has remained free of recurrences since moving to a warmer climate. The changes were early in her case, permitting restoration of 20/15 vision in the only eye affected. It seems at least possible that the vascular condition was of importance in this case.

The second patient was a man of 43, who showed typical periphlebitis retinae, with perivascular sheathing, closure of a number of retinal veins and pre-retinal hemorrhages which organized and showed a growth of new vessels into them. The tuberculin tests were positive, but after a long course of tuberculin, when he was tuberculin-negative, fresh areas of periphlebitis continued to develop. He was referred to Doctor de Takats, who found evidence in the extremities of Buerger's disease. There was absence of pulsation in one dorsalis pedis artery and other circulatory changes. A heparin clearance test showed a flat curve of the type seen in the active stage of Buerger's disease. He gave up smoking, was given injections of sodium tetrathionate and later was kept on small doses of dicumarol by mouth. There have been no fresh hemorrhages for some time, and central vision has returned to normal.

The three patients in whom the vascular condition seemed the only cause of the condition deserve a more detailed report. The first was a girl of 23 with the picture of unilateral Eales' disease of several years standing with recent hemorrhages. Tuberculin tests were negative as were other examinations with the exception of the pressor tests which showed marked angiospasm. She was treated with barbiturates and tissue extract and showed no further hemorrhage during observation. Vision, which was reduced to light perception, showed no improvement but the other eye remained normal.

The second patient, Mary D., was seen at the age of 12, showing retinal hemorrhages and bands of organizing connective tissue along the vessels about the discs. No evidence of tuberculosis or other infections was revealed, but pressor tests showed marked angiospasm, with a drop of temperature following nicotine amounting to 13° F. Hemorrhages continued to occur for a time in spite of antispasmodic therapy, and it became impossible to see either fundus. These hemorrhages have finally ceased, and vision in one eye, after removal of the lens which had become cataractous, is 20/200. The other eye has developed a cataract and has faulty projection.

The third patient, Joseph L., was a boy of eight. He was first seen when repeated hemorrhages had reduced his vision to: right, light perception; left, 6/200. He showed marked signs of peripheral angiospasm. The temperature at the toes was low and rose 10.5° F. after nerve block. Oscillometric readings were low. Other examinations, including tuberculin tests, were negative. On treatment with prostigmine, 7½ mg. t.i.d., he remained free from further hemorrhages for a year with marked improve-

ment in vision. One hemorrhage occurred at this time but after resuming treatment, vision continued to improve and at the end of two years was: right, 20/200; left, 20/65.

On looking over the material it is plain that the situation is by no means so simple as in central angiospastic retinopathy. In periphlebitis retinae we must admit that several factors may be responsible and that it is by no means easy to determine from the clinical picture which of these is the most likely one. In some cases, and perhaps the majority, we must consider that tuberculosis is of causative importance. In others, it must be said that no cause can be found. In a small group, however, the factor of peripheral vascular disease seems to be of primary importance. Even in these cases, the relationship of spasm to the fundus changes it produces is by no means so definite as in central angiospastic retinopathy. We must assume, as in Buerger's disease, that chronic or repeated angiospasm causes organic changes, including, aside from ischemia of the tissues supplied, stasis in the veins, with perivascular exudation and all the subsequent changes which one sees in the extremities. In fact, for this limited group of cases, the term thrombo-angiitis obliterans employed by Marchesani would seem to be appropriate. Aside from spasm, there may be, as in one case mentioned above, anomalies in the clotting mechanism, as shown by heparin clearance and other tests. While the results of treatment are by no means so favorable as in central angiospastic retinopathy, the complete resistance of this much more serious condition to all forms of therapy in the past gives such results as have been reported in these few cases at least a certain interest.

It would certainly seem that in such a serious condition, obviously involving the retinal vessels, examination of the peripheral vascular system should be included in the complete examination of every case. Cases showing positive findings, even when tuberculin tests are also positive, may well be given the benefit of treatment designed to improve the peripheral circulation.

There are other ocular conditions in which peripheral angiospasm undoubtedly plays a part. Some of these, including spasm of the central retinal artery, have been discussed in the previous report. There is time here to speak of only one other condition, illustrated by a single case. Mrs. K., a woman of 53, was operated upon for senile cataract of the left eye in December, 1941. The usual preliminary tests revealed normal findings. Following an uneventful intracapsular extraction, vision of 20/20 was obtained. After six months, however, vision began to fail, with no fundus changes except a gradually progressive diminution in the size of the retinal arterioles. After two months of treatment with thiamin and B complex in large doses, vision was reduced to 20/80. At this time the patient was hospitalized for examination and treatment by Doctor Van Dellen. He reported a high degree of peripheral angiospasm, and treatment with prostigmine and tissue extract was begun. Vision began to improve slowly, and within four months it was 20/40. The arteries were still rather small. The right eye was operated upon at this time, and a vascular condition similar to that in the left eye was revealed, with best obtainable vision 20/65. Prostigmine was resumed and two further courses of tissue extract were given. Vision slowly improved in both eyes, and 15 months after the first operation it was: right, 20/30; left, 20/20. The vessels of the right eye now show a definite increase in size, being practically normal, while the disc which

had been pale has improved slightly in color. The vessels of the left eye are still small, but not so small as they had been at the height of the disturbance. In this case, the only one in my experience, there seemed to be no doubt that arteriolar spasm was responsible for the loss of vision and that antispasmodic treatment affected the outcome favorably. The difficulty of judging the size of the retinal vessels in aphakic patients is admitted, but when fundus examination reveals no other cause for loss of vision in such cases, or in other cases, and the retinal arterioles appear small, it would seem logical to advise examination of the peripheral vascular system.

CONCLUSIONS

1. Additional evidence is presented from the records of 23 cases that peripheral angiospasm is the cause of what has been described as central angiospastic retinopathy.
2. The status of periphlebitis retinae with recurring vitreous hemorrhages in young adults is reviewed, and evidence is presented that in a certain proportion of such cases peripheral vascular disease is the primary cause.
3. A case is presented of extreme attenuation of the retinal arterioles in a postoperative cataract patient who showed marked peripheral angiospasm and improved on antispasmodic treatment.
4. It is suggested that in all cases showing the characteristics of these three syndromes a careful examination of the peripheral vascular system is indicated. In other conditions in which it is desired to employ vasodilators it would seem logical to advise such an examination before treatment is begun.

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TRANSACTIONS

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High-Lights of the Forty-Seventh Annual Convention

The American Academy of Ophthalmology and Otolaryngology held its 48th annual convention at the Palmer House, Chicago, October 10-13, 1943.

Officers Elected — 1944

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Detroit, Michigan.

Chicago — 1944

Chicago has been selected by the Council as the city where the 1944 convention will be held. The final decision, however, as to date and place, is to be left to the discretion of the Program Committee at its January meeting because of possible unforeseen developments in the war situation.

Registration of Members and Guests

Registration, inclusive of members, guests, exhibitors and ladies, totaled 1,945.

President's Reception

On Sunday afternoon, the president's reception was attended by more than 700 members and guests. In the receiving line were Dr. and Mrs. James A. Babbitt, Dr. and Mrs. Ralph A. Fenton, Dr. and Mrs. Lawrence T. Post and Dr. and Mrs. Sanford R. Gifford.

Honor Society

The following men were elected to the Honor Society of the Academy: Dr. Gabriel Tucker, Philadelphia; Dr. A. W. Proetz, St. Louis; Dr. Perry Goldsmith, Toronto, Canada; Dr. George M. Coates, Philadelphia; Colonel J. E. Ash, Washington, D. C.

Dr. Thomas E. Carmody, Denver, was elected president of the Honor Society. He will take office January 1, 1944. Dr. Frank E. Burch, St. Paul, was named president-elect. Members of this society are elected in recognition of their scientific achievements and of their services to the Academy.

Doctor Tucker is professor of bronchology and esophagology at the University of Pennsylvania and he is professor of bronchology, esophagology and laryngeal surgery at the Graduate School, University of Pennsylvania. He is a past chairman of the section of laryngology, otology and rhinology of the American Medical Association. Doctor Proetz is professor of clinical otolaryngology at Washington University, St. Louis. He was first vice president of the Academy in 1940, vice president of the American Laryngological Association in 1941 and chairman of the section of otolaryngology of the American Medical Association in 1940. Doctor Goldsmith is professor emeritus of the department of otolaryngology at Toronto University. He was Guest of Honor at the 46th annual meeting of the Academy in 1941. He is a Colonel in the medical corps of the Royal Canadian Army. Doctor Goldsmith is a member of the leading otolaryngological societies of the United States and is a past president of the American Laryngological, Rhinological and Otological Society. Doctor Coates is professor emeritus of the department of otolaryngology at the University of Pennsylvania; professor of otorhinology at the Graduate School of the University of Pennsylvania; Colonel in the medical reserve corps of the U. S. Army; past president of the Academy (1939). He was secretary of the American Laryngological Association, 1920-32; president of the American Laryngological Association, 1933; president of the American Laryngological, Rhinological and Otological Society, 1935-1936; president of the American Otological Society, 1941; chairman of the section of otolaryngology of the American Medical Association, 1926. Colonel J. E. Ash is from the Surgeon General's Office in Washington, D. C. He is curator of the Army Medical Museum and registrant of the Registry of Otolaryngic Pathology and Ophthalmic Pathology. He is a co-author of the Atlas of Otolaryngic Pathology and the Atlas of Ophthalmic Pathology which is sponsored and distributed by the American Academy of Ophthalmology and Otolaryngology.

Scientific Programs

The joint session was held on Sunday afternoon, October 10, at two o'clock. President James A. Babbitt spoke on "Our Academy"; Dr. Ralph A. Fenton, Guest of Honor, discussed "The Unity of Medical Science in the Americas," and a symposium entitled "Seeing, Hearing, Thinking and Doing — A Problem of American Youth" was presented by Dr. Joseph E. Raycroft, Princeton, New Jersey (by invitation), Dr. A. D. Ruedemann and Dr. A. C. Furstenberg.

The scientific programs were continued in the afternoon sessions, Monday, Tuesday and Wednesday, according to the schedule outlined.

Inter-American Relations Dinner and Program

The Inter-American Relations Dinner and Program was held Tuesday evening, October 12, in the Grand Ball Room of the Palmer House. President James A. Babbitt gave an address of welcome and he introduced Dr. Ralph A. Fenton, Guest of Honor, who was presented an award of merit which consisted of a plaqued certificate. The president-elect, Dr. Lawrence T. Post, was introduced by Dr. William L. Benedict. Dr. Harry S. Gradle introduced the invited ophthalmologists of Latin America and Kellogg Foundation Fellows of Latin America, Dr. Chevalier L. Jackson the invited otolaryngologists of Latin America and Dr. Walter H. Theobald the Latin American Consuls residing in Chicago. The speakers of the evening were Colonel Albert Dreisbach who discussed "General Survey of the Activities of Our Division in Latin America," and Dr. William Lytle Schurz whose subject was "Cultural Relations and the Good Neighbor Policy." Dinner music was furnished by a string ensemble. The Great Lakes Naval Training Station Fifty Voice Choir, under the direction of Lt. Comdr. Hjalmer Hanson, also furnished music throughout the evening.

Dr. Gordon B. New President-Elect

Dr. Gordon B. New, professor of laryngology and oral and plastic surgery, Mayo Foundation Graduate School of the University of Minnesota, and head of the section on oral and plastic surgery at the Mayo Clinic, Rochester, Minnesota, was named president-elect of the Academy at its annual business session Tuesday, October 12.

Doctor New received his medical degree from Toronto University. He has been associated with the Mayo Clinic since 1910. He has been certificated by the American Board of Otolaryngology and the American Board of Plastic Surgery, and is a member of the American Association of Oral and Plastic Surgery, American Laryngological Association and the American Laryngological, Rhinological and Otological Society.

Dr. Lawrence T. Post, professor and head of the department of clinical ophthalmology at Washington University, St. Louis, is the present president-elect. He will take office January 1, 1944, and Doctor New will become president January 1, 1945.

The following secretaries were re-elected: Dr. William L. Benedict, executive secretary-treasurer; Dr. O. E. Van Alyea, secretary for otolaryngology, Dr. Algernon B. Reese, secretary for ophthalmology; Dr. Dean M. Lierle, secretary for instruction in otolaryngology and maxillofacial surgery; Dr. A. D. Ruedemann, secretary for instruction in ophthalmology; Dr. Erling W. Hansen, secretary for public relations. Dr. Harry S. Gradle was elected secretary for home study courses. Dr. James M. Robb was elected councillor for a term of four years.

Scientific Exhibit Awards

The Academy presented six awards to participants in its scientific exhibit in connection with the annual session. Three awards went to exhibits on otolaryngology and three to exhibits on ophthalmology.

Dr. A. D. Ruedemann, Cleveland, received the award for originality in oph-

thalmology on his exhibit entitled "Visual Field Changes—Region of Chiasm." Dr. J. R. Lindsay, Chicago, exhibitor, and Dr. H. B. Perlman, Chicago, co-exhibitor, received the award for originality in otolaryngology on their exhibit entitled "Meniere's Disease" and "Eye Movements." The award for greatest value to medicine in ophthalmology was given to Dr. Roy O. Riser, Chicago, for his exhibit showing a review of accepted and recently introduced eye medications. The award for greatest value to medicine in otolaryngology was given to the exhibit sponsored by the Committee on the Conservation of Hearing, under the direction of Dr. Horace Newhart, Minneapolis, which presented recent information regarding the discovery and care of hearing deficient school children. For the exhibit displaying conditions of unusual interest in the Registry of Ophthalmic Pathology, Col. J. E. Ash, Washington, D. C., exhibitor, and Helenor Campbell Wilder, Washington, D. C., co-exhibitor, received the award for excellence in presentation in ophthalmology.

The award for excellence in presentation in otolaryngology was presented to Dr. Samuel Fomon, New York, exhibitor, Dr. Romeo A. Luongo, Philadelphia, co-exhibitor, Dr. Morris S. Bender, New York, co-exhibitor, and Dr. Alfred Schattner, New York, co-exhibitor. The display consisted of a series of fifty diagrams showing stages of typical rhinoplastic operation, plaster casts demonstrating the technic, x-ray films depicting recent and old unreduced nasal fractures and motion pictures showing the role of rhinoplasty in restoration of function.

Kellogg Fellows

The Academy was host to nine of the Latin American doctors who are now in this country on Pan-American Congress of Ophthalmology-W. K. Kellogg Foundation Fellowships. At a luncheon meeting on Wednesday, October 13, they formed the "Sociedad de Los Kellogg Fellows" and made Dr. Harry S. Gradle Honorary President. The purpose of the Society is to maintain contact between present and future Kellogg Fellows, to endeavor to help select future men for Kellogg Fellowships, and to try to establish organized graduate teaching after they return to their respective countries.

PROGRAM COMMITTEE MEETING

The Program Committee will meet in Chicago in January, 1944. Requests for participation in the scientific program and other branches of the Academy's program should be in the hands of the respective secretaries before January.

O. E. Van Alyea, M.D., Chicago, Secretary for Otolaryngology — scientific papers, motion pictures and scientific exhibits.

Algernon B. Reese, M.D., New York, Secretary for Ophthalmology — scientific papers and motion pictures.

THE PAN-AMERICAN CONGRESS OF OPHTHALMOLOGY

Inasmuch as documentary evidence of the origin of the Pan-American Congress of Ophthalmology is so scattered that a compilation in future years would present enormous difficulties, it would not seem amiss at this time to review the various causes and events that led to the formation and development of this forum of scientific exchange. The complete upset of existing international media for the dissemination of knowledge and discussion of events in scientific ophthalmology is of course due to the World War and judging from the experiences subsequent to the First World War, much time will elapse before the old established paths are renewed.

The International Congress of Ophthalmology was initiated by Dr. Worlemont of Brussels where the first meeting was held in 1857. It was a quadrennial affair and regular meetings were held every four years up to 1914. The meeting of that year was scheduled for St. Petersburg, Russia, and all plans were laid to assemble in August of that year. These were of course nullified by the outbreak of World War I. Owing to the resultant bitter chauvinism, regular meetings could not be resumed subsequent to the signing of the Peace Treaty. But the need for an international forum was felt and through the efforts of some of the American and British leaders, a Congress of English speaking ophthalmologists was held in Washington in 1922 and a similar one in London in 1925. Due to these efforts, the International Congress was resumed and the most successful meeting of all time was held in Amsterdam in 1929. Subsequent meetings took place in Madrid in 1933 and in Cairo in 1937. Vienna was chosen as the site for the 1941 meeting and the Council which was composed of a representative from each of the major countries of the world, meeting in London in the Spring of 1939, confirmed the site of meeting, despite the fact that war was imminent and the blunt statement that none of the ophthalmologists from what are now known as the Allied Nations would attend such a meeting in such a place. It then became evident that there would be no International Congress of Ophthalmology in 1941 and probably not for many subsequent years.

Then was conceived the idea of a Congress of Ophthalmology of the Western Hemisphere alone, to which, of course, could be given only the name Pan-American Congress of Ophthalmology. The original correspondence was started in May of 1939 between Dr. M. Alvaro of Sao Paulo, Brazil, and Dr. H. S. Gradle of Chicago. A working plan was evolved which required the moral and financial support of a strong ophthalmological society and the official backing of the Department of State. When the plan was presented to the Council of the American Academy of Ophthalmology and Otolaryngology, it was quickly adopted. A committee was appointed and given adequate financial support to initiate proceedings as rapidly as possible. To this committee were appointed Dr. H. S. Gradle, chairman, Chicago, Illinois; Dr. Conrad Berens, New York, N. Y.; Dr. Moacyr Alvaro, Sao Paulo, Brazil, all of whom are members of the Academy.

The original plan, which was carried through with but comparatively few changes, called for the first meeting of the PACO, both organizational and scientific, to be held in Cleveland, Ohio, immediately following the annual meeting of the American Academy of Ophthalmology and Otolaryngology in 1940. Less than eighteen months were available for the necessary planning and publicity. But thanks to the unswerving and speedy help of the Academy and of the Department of State of the U.S.A., plans materialized in a rapid and smooth sequence of events.

The first and probably the most difficult aspect was the notification of all ophthalmologists of the Western Hemisphere of the formation of the new Congress. For this purpose, notice appeared in all of the specialty journals, not once, but in nearly every issue. The general medical journals of national and international circulation contributed their share to the publicity campaign, as did some of the non-medical scientific organs. Up and down the length and breadth of the Western Hemisphere, the word went forth that ophthalmology was about to give birth to a new public forum for exchange of scientific views, a forum that would not be interrupted by the jealousies of war and chauvinism, and one in which the rapidly progressing

science of the New World would find free expression, unhampered by the paternalism so dominant in the old International Congress of Ophthalmology.

But printed publicity alone was not sufficient to reach into all the nooks and crannies of this hemisphere. The ophthalmologists of Canada and of the United States were well organized and could be reached through the printed columns of medical publications without too great difficulty. But the same did not hold true with the colleagues in all of the Latin-American Republics. By unknown methods that seemed to partake of ancient alchemy, Dr. Alvaro was able to produce a fairly accurate list of ophthalmologists south of the Rio Grande. To each of them was sent a personal formal invitation to participate in the coming Congress. These engraved invitations were produced through the generous contributions made by the Bausch & Lomb Optical Company, the American Optical Company, and the Abbott Laboratories. They were forwarded by the Department of State.

At this point, it would seem most appropriate to pay tribute to the generous help given by the Department of State, especially that of Mr. Charles Thomson, Chief of the Division of Cultural Relations. Inasmuch as the Congress of the United States never passed legislation that recognized the formation or existence of the PACO or authorized expenditures of funds for that organization, the Department of State was able to extend only non-financial help. But without the wise guidance of the Department, the PACO would have fallen into many a pitfall that would ultimately have jeopardized its existence.

Then a list was compiled of all Latin-American ophthalmological societies, both local and national, all universities and clinics in which there were ophthalmologists, and such other organized medical groups as were interested in ophthalmological work. These were invited to appoint representatives to the coming Congress, as well as to urge their individual members to attend. And finally the Department of State invited the governments of the other American Republics to appoint official ophthalmological representatives to the Congress.

In order to have as large a representation of ophthalmologists as possible at the meeting, it was decided to have the PACO meet on October 10-12, 1940, in the Hotel Cleveland in Cleveland. While it was realized that this location was some distance from the Atlantic seaboard, where the majority of the Latin-Americans would disembark, it was thought advantageous to utilize the presence of a large number of ophthalmologists of the United States who would convene in Cleveland for the preceding five days for the annual meeting of the A.A. of O. & O. The twelve hours additional travel from New York could play but little role to men who had already journeyed from twelve to twenty days to reach this country, and the correctness of this reasoning was shown by the results.

A tentative plan of organization of the Congress was drawn up by the committee appointed by the A.A. of O. & O., all of whom had had extensive experience with the management of national as well as international medical organizations. Until the actual meeting of the Congress, all of the spade work was performed consistent with that plan which in due time was presented to the Congress and was adopted with but slight modifications. The committee was organized with Dr. H. S. Gradle as chairman, Dr. C. Berens as secretary and treasurer for the lands north of the Panama Canal, and Dr. M. Alvaro as secretary for all that lay south of the Canal. The program for the meeting was developed by the two secretaries on the basis of dividing the number of papers equally between the countries of the Northern and the Southern Hemispheres. Tri-lingual programs in Spanish, Portuguese, and English were printed, bearing the newly adopted seal of the PACO. This consisted of an embossed circle surrounding an outline map of the Western

Hemisphere. At the four cardinal points around the circle were the letters P A C O which stand for Pan-American Congress of Ophthalmology in any one of the three official languages.

An invitation had been issued to all Latin Americans to be the guests of the

Academy for the annual Academy meeting, which by previous arrangement partook of a Pan-American character. The presidential address by Dr. Frank E. Brawley of Chicago dealt with the development of the PACO and how necessary it had become through the death of the International Congress of Ophthalmology. The annual banquet of the Academy was strictly Pan-American with flags of all the American Republics festooning the tables and the speeches featuring the Western Hemispheric goodwill. Throughout the week a special information booth was maintained with Spanish speaking clerks and at this booth ophthalmologists registered for admission to the PACO. The committee had decided to admit any reputable ophthalmologist to membership, waiving specific qualifications because of differences that exist in the medical conditions among the various American Republics. A registration fee of five dollars was charged and all members were given an attractive badge, designed around the official seal of the PACO.

Much thought and preparation was given to means of overcoming language difficulties anticipated during the progress of the scientific meeting. Due to the ingenuity and intensive preliminary work of the two secretaries, that difficulty became non-existent. Every paper had been abstracted and condensed to some ten or twelve paragraphs weeks previous to the meeting. These paragraphs from Spanish and Portuguese papers were then translated into English and the English papers into Spanish or Portuguese. The abstracts were then put on lantern slides and projected as the paper was being presented in the original language. Thus all members were enabled to follow the context of the papers regardless of the language, and could discuss the papers in their own language. The discussion of each paper was opened by a previously appointed member, English speaking ophthalmologists discussing the papers from Latin America and vice versa. The scheme proved to be most satisfactory and elicited much praise from members of all nationalities.

The program was as follows:

FRIDAY, OCTOBER 11, 1940

1. Value of Gonioscopy in the Prognosis and Treatment of Glaucoma.
   By Manuel Uribe Troncoso, New York City
   Discusser: H. S. Sugar, Chicago, Illinois
2. A Mathematical Interpretation of Field Acuity
   By Albert C. Snell and Scott Sterling, Rochester, New York
   Discusser: Herman M. Burian, Hanover, New Hampshire
3. Orbital Hyperostosis
   By William L. Benedict, Rochester, Minnesota
   Discusser: A. D. Ruedemann, Cleveland, Ohio
4. Ocular Leprosy
   By Jose Mendonca de Barros, Sao Paulo, Brazil
   Discusser: Peter C. Kronfeld, Chicago, Illinois
5. Keratoconjunctival Lesions Prevalent in the High Altitudes of Bolivia
   By Aniceto Solares, Sucre, Bolivia
   Discusser: Edward Jackson, Denver, Colorado
6. Hyphema after Cataract Operation
   By Derrick T. Vail, Cincinnati, Ohio
   Discusser: Horacio Ferrer, Havana, Cuba
7. Observations on the Fundus Oculi in Diabetes Mellitus
   By S. Hanford McKee, Montreal, Canada
   Discusser: Jose Mendonca de Barros, Sao Paulo, Brazil
8. Hypertonic Rigidity of Convergence of False Paralysis of Divergence
   By A. Vasquez-Barriere, Montevideo, Uruguay
   Discusser: Avery D. Prangen, Rochester, Minnesota
9. Dacryocystorhinostomy
   By Tomas Yanes, Havana, Cuba
   Discusser: Ralph O. Rychener, Memphis, Tennessee
10. Unusual Macular Lesions Illustrated with Colored Fundus Photographs
    By Arthur A. Bedell, Albany, New York
    Discusser: Heitor Marback, Bahia, Brazil.

SATURDAY, OCTOBER 12, 1940

11. The Best and Most Exact Method of Diagnosing Small Astigmatic Errors by Means of Bicylindrical Combinations
    By Manuel Marquez, Madrid, Spain
    Discusser: William H. Crisp, Denver, Colorado
12. Interstitial and Ulcerative Keratitis in Leishmaniasis
    By Cesario de Andrade, Bahia, Brazil

Discusser: Phillips Thygeson, New York City

13. Complications of Cataract Removal and Measures that may be employed for their Relief
By John O. McReynolds, Dallas, Texas
Discusser: Penido Burnier Filho, Sao Paulo, Brazil

14. Intra-Capsular Extraction of Cataract with a New Vent
By Horacio Ferrer, Havana, Cuba
Discusser: Theodore J. Dimitry, New Orleans, Louisiana

15. Further Observations on the Nature, Etiology, and Significance of Exfoliation of the Lens Capsule
By Rodman Irvine, Los Angeles, California
Discusser: Arturo Quevedo, Guatemala City, Guatemala

16. Ocular Filiarosis
By Arturo Quevedo, Guatemala City, Guatemala
Discusser: Nelson M. Black, Miami, Florida

17. Chemotherapy in Ophthalmology
By Alan C. Woods and Jack S. Guyton, Baltimore, Maryland
Discusser: Genserico Gonzaga Jayme, Annapolis, Goyas, Brazil

18. Toxic Syndrome of Ocular Tuberculosis
By Carlos Charlin, Santiago, Chile
Discusser: Albert L. Brown, Cincinnati, Ohio

19. Further Reports on Tendon Transplantation for Abducens Paralysis
By Sanford R. Gifford, Chicago, Illinois
Discusser: Tomas R. Yanes, Havana, Cuba

20. Tumors of the Optic Nerve
By J. Pereira Gomes, Sao Paulo, Brazil
Discusser: Frederick A. Davis, Madison, Wisconsin

21. Congenital Bilateral Colobomata of the Upper and Lower Eyelids. Surgical Correction by Sliding Pedicle Flap
By Daniel B. Kirby, New York City
Discusser: Colombo Spinola, Bahia, Brazil

22. Use of the Sodium Light in Examining the Fundus of the Eye
By Justo Lijo Pavia, Buenos Aires, Argentina
Discusser: Alfred C. Cowan, Philadelphia, Pennsylvania

23. Cephalic Tetanus Following Ocular Injury
By Genserico G. Jayme, Annapolis, Goyas, Brazil
Discusser: Virgil G. Casten, Boston, Massachusetts

24. The Conjunctiva as an Entrance of Infection in Chagas' Disease (Trypanosomiasis Americana)
By Cesario de Andrade, Bahia, Brazil
Discusser: Charles A. Bahn, New Orleans, Louisiana.

The meeting was attended by approximately 240 members from the United States and Canada and 25 from the Latin-American Republics. In view of the comparatively short notice and of the conditions resulting from the war in Europe, this attendance was considered to be most satisfactory. On Friday evening, October 11th, there was an informal dinner which the majority of the members attended, accompanied by their wives. Short speeches were the order of the day, the majority being given by the Latin Americans upon specific request.

The business meeting on the subsequent day took something less than an hour. First came a report of the Organizing Committee, which was approved, and their proposed general plan of operation was accepted as the basis for the continuation of the PACO. The presentation and acceptance of a formal constitution was reserved for some future meeting. It was decided that there should be a president, seven vice-presidents, and two secretaries, one of whom should be the treasurer. The following were elected:

President

Dr. H. S. Gradle
58 East Washington Street
Chicago, Illinois, U. S. A.

Vice-Presidents

Dr. S. H. McKee
Montreal, Canada

Dr. F. Brawley
Chicago, Illinois, U.S.A.

Dr. E. Jackson
Denver, Colorado, U.S.A.

Dr. T. Yanes
Havana, Cuba

Dr. E. Demaria
Buenos Aires, Argentina

Dr. J. Pereira Gomes
Sao Paulo, Brazil

Dr. A. Vasquez-Barriere
Montevideo, Uruguay

Executive Secretaries

Dr. M. Alvaro
1151 Rua Consolacao
Sao Paulo, Brazil

Dr. C. Berens (Treasurer)
477 First Avenue
New York, New York, U.S.A.

Subsequently Professor Demaria of Buenos Aires felt that his other obligations prevented his acceptance of the position and in his place was elected Dr. F. Belgeri of the same city. In the winter of 1942-43 the PACO suffered the loss by death of Dr. S. Hanford McKee of Montreal and Dr. Edward Jackson of Denver, both vice-presidents. Their places were filled by Dr. John A. MacMillan of Montreal and Dr. F. C. Cordes of San Francisco.

It was decided to form a Council to consist of one member from each one of the Republics, regardless of the numerical membership from the country. In each country the Council member was to be elected by the national ophthalmological society or societies, and in those countries in which such an organization did not exist, he was to be appointed formally by the government of the country. Following is the present membership of the Council:

ARGENTINA—Dr. Esteban Adrogue, Buenos Aires
BOLIVIA—Dr. Aniceto Solares, Sucre
BRAZIL—Dr. Moacyr E. Alvaro, Sao Paulo
CANADA—Dr. C. A. Campbell, Toronto
CHILE—Dr. Santiago Barenechea, Santiago
COLOMBIA—
COSTA RICA—Dr. Alexis Aguero, San Jose
DOMINICAN REPUBLIC—Dr. Wenceslao Medrano Fo, Ciudad Trujillo
CUBA—Dr. R. Hernandez Arias, Havana
ECUADOR—Prof. Jose Meguel Varas Samaniego, Guayaquil
GUATEMALA—Dr. R. Pacheco Luna, Guatemala City
HAITI—Dr. Elie Villard, Port-au-Prince
HONDURAS—
MEXICO—Dr. Rafael Silva, Mexico City
NICARAGUA—
PANAMA—Dr. Carlos N. Brin, Panamanian Embassy, Washington, D. C.
PARAGUAY — Dr. Gustavo Vasquez, Asuncion
PERU—Dr. Jorge Valdeavellano, Lima
SALVADOR—
URUGUAY—Dr. Carlos M. Berro, Montevideo
UNITED STATES—Dr. William L. Benedict, Rochester, Minnesota
VENEZUELA—Dr. J. M. Espino, Caracas

Membership was opened to any ethical ophthalmologist of the Americas. It was decided that the meetings should be held every three years, alternating as far as possible between the Northern and Southern Hemispheres, and the next meeting was scheduled for Montevideo, Uruguay, in November 1943. For that and subsequent meetings, a local committee was to be appointed to arrange local details and to cooperate with the officers of the PA CO in the selection of the scientific program. The official languages of the Congress were designated as English, Portuguese, and Spanish. Guests whose mother-tongue was one of these three would always be welcomed, but ophthalmologists of other nationalities would require special invitation. It was further decided that work in the inter-convention periods was to be carried on by the officers and by special committees.

The scientific session was resumed after the business meeting and carried through to a successful conclusion. The meeting was adjourned about five o'clock on Saturday evening. The Latin-American members subsequently visited many of the American ophthalmological clinics, particularly in the eastern parts of the United States. The consensus of opinion was that the meeting had been a successful scientific venture, that the Pan-American Congress of Ophthalmology was an assured organization, and that all efforts would be made to enlist hemisphere-wide cooperation for its future development.

However, the financial situation of the PACO was rather precarious. The ground-work and the first meeting were financed exclusively by the Academy, except for the gift of $600.00 by the American Optical Company, Bausch & Lomb Optical Company, and the Abbott Laboratories. That left practically nothing for the PACO to work with. So a membership campaign was initiated by postal card solicitation and by notices published in the ophthalmological journals. To those contributing $5.00 per year, in addition to

membership in the PACO, was sent an annual subscription to Ophthalmologia Ibero - Americana, the quarterly abstract journal founded and published by Dr. M. Alvaro in Sao Paulo. Thus sufficient funds became available to carry on the work until the time of the next meeting which is to be financed by a registration fee from all members attending and from the sale of exhibition space to the commercial exhibitors.

Several committees were appointed at or subsequent to the Cleveland meeting. Their activities have been so dependent upon extraneous circumstances that some have accomplished a great deal whereas others have been forced to remain idle through no fault of their own. It might be well to discuss each committee individually at somewhat greater length.

It was soon realized that for a freer exchange of scientific information and thought between the ophthalomolgists of the United States and those of the Latin-American Republics, more uniform terminology was necessary. This could be obtained only through the existence of a uniform textbook of ophthalmology printed in both English and Spanish. The texts used in Latin-America were reviewed, but it was soon discovered that there was no universally accepted textbook of ophthalmology in either Spanish or Portuguese. But in the United States "The Eye and Its Diseases," a cooperative book written by many leading ophthalmic authorities and published by W. B. Saunders & Co., under the editorship of Dr. Conrad Berens, has attained universal recognition. Doctor Berens and Doctor Alvaro are now arranging for the publication of that text in Spanish. Each chapter is being translated and revised to date by an outstanding Latin-American ophthalmologist and the editing will be done by the committee. A publishing house in South America has concluded financial arrangements with the Saunders Company and it is expected that the book will appear within a comparatively short time. Thus ocular terminology will be more or less codified throughout the Western Hemisphere whose oculists will then be reading and speaking the same scientific language. The Pan-American Congress of Ophthalmology is the sponsor of the work.

The Committee on the Exchange of Literature is headed by Dr. J. Lijo Pavia of Buenos Aires, Argentina, and has as members the following:

Dr. Moacyr E. Alvaro, Sao Paulo, Brazil
Dr. Raul Arganaraz, Buenos Aires, Argentina
Dr. W. L. Benedict, Rochester, Minnesota, U.S.A.
Dr. Penido Burnier, Campinas, Brazil
Dr. A. Torres Estrada, Mexico City, Mexico
Dr. Morris Fishbein, Chicago, Illinois, U.S.A.
Dr. W. B. Mattos, Sao Paulo, Brazil
Dr. Mesquita Sampaio, Sao Paulo, Brazil
Dr. A Riquelme, Asuncion, Paraguay
Dr. H. Rocha, Bello Horizonte, Brazil
Dr. B. Samuels, New York City, U.S.A.
Dr. P. Seremina, Montevideo, Uruguay
Dr. C. Spinola, Bahia, Brazil
Dr. Derrick Vail, Cincinnati, Ohio, U.S.A.
Dr. Weskamp, Rosario, Argentina
Dr. T. Yanes, Havana, Cuba

Owing to the wartime restrictions, it has not been possible for this committee to effect the literary exchanges between the various Republics that it had planned. Nor is it likely that much will be accomplished until normal transportation is reestablished.

The Committee on Professorial Exchange is under the chairmanship of Dr. Louis J. Fernandez of Puerto Rico, and numbers as its members:

Dr. Cesario de Andrade, Rio de Janeiro, Brazil
Dr. Raul Arganarez, Buenos Aires, Argentina
Dr. J. M. Espino, Caracas, Venezuela
Dr. Carlos Charlin, Santiago, Chile
Dr. Sanford R. Gifford, Chicago, Illinois, U.S.A.
Dr. Rafael Silva, Mexico City, Mexico
Dr. Alan C. Woods, Baltimore, Maryland, U.S.A.

Tentative plans for the exchange of teachers (Visiting Professorships) were discussed by this committee, but were laid aside because of the lack of funds and because of the impossibility of extramural travel.

The Committee on the Prevention of Blindness is most active under the dynamic leadership of Dr. F. Belgeri of Buenos Aires, whose efforts are so ably seconded by the secretary, Dr. B. Courtis, also of Buenos Aires. The other members of that committee are:

Dr. E. C. Ellett, Memphis, Tennessee, U.S.A.
Dr. H. Ferrer, Havana, Cuba
Dr. Natalicio Farias, Rio de Janeiro

A questionnaire has been sent to all of the Republics of the Western World, aimed at a compilation of statistics on the causes of blindness, the number of blind, the legislative status of prevention of blindness, the agencies both governmental and private with that objective, and the numerous other aspects that are concerned with that problem. The committee will collect those statistics into an exhaustive report that will be presented at the next meeting of the PACO for which a full half day has been set aside in the program. It is anticipated that much will be accomplished by this report, particularly in the initiation of legislation required to lessen the scourge of blindness.

The Committee on Trachoma is performing an equally important function. Throughout the Western Hemisphere, that dread disease has spotted certain areas and left others untouched, and our knowledge of the location and extent of the infected areas leaves much to be desired. Dr. Ivo Correa Meyer of Porto Alegre, Brazil, is the chairman of this important committee, which is composed of:

Dr. J. Sena, Buenos Aires, Argentina
Dr. J. Valdeavellano, Lima, Peru
Dr. Phillips Thygeson, New York City, U.S.A.
Dr. Polk Richards, U.S.A.

A comprehensive questionnaire has been sent to all countries and reports are now being received, so that it will be possible in the near future to prepare a spot map of Trachoma in the Americas. With the modern methods of treatment and the restriction of migration to and fro from infected areas, governmental sanitarians should be able to drive this disease out of the Western Hemisphere within a few generations, after they have been shown where it is and how much of it there is. For the presentation of this report and the consequent discussion, a half day has been set aside during the coming meeting of the PACO.

A very necessary adjunct for the development of any organization is a good "house organ." The PACO was in no position to establish another ophthalmological journal, but in casting around the field, it was found that a comparatively new magazine with a limited circulation in South America could be adapted to the purpose. So arrangements were concluded that Ophthalmologia Ibero - Americana should become the official journal of the PACO. This quarterly abstract journal was published in Brazil under the editorship of Dr. M. Alvaro, who was willing to expand it to meet the requirements of an international organization. Owing to the lower costs of publishing in Brazil, a gratis subscription could be given to all members of the PACO, from whose membership dues two dollars were sent to Ophthalmologia. In return, the magazine became tri-lingual; that is, every article, every abstract, and every news item appears in either English and Spanish or Portuguese and English. Pages are devoted to items of international interest so that all members are informed constantly of Pan-American developments in the field of ophthalmology. Unfortunately, the first two issues were lost in transit to this country through submarine warfare and as a result the ophthalmologists of the United States are not yet as familiar with Ophthalmologia as they will be. It might be noted here that the American Journal of Ophthalmology is now devoting a special section to Pan-American news, under the direciton of Dr. Uribe Troncoso, formerly of Mexico, but now a resident of New York City.

In ophthalmological circles there are recognized undergraduate, graduate, and postgraduate students. The first is of course the medical student who has not yet finished his career in the medical school; the second is a graduated physician who is spending from one year upward in an ophthalmological institution in preparation for practice in ophthalmology; and the third is a practicing ophthalmologist who is taking special courses or work to further his knowledge in some particular aspect of the specialty. From the time of its inception, it had been one of the aims of the PACO to further the exchange of graduate students between the Americas. During the course of an informal conversation, Dr. George Darling, the president of the W. K. Kellogg Foundation of Battle Creek, Michigan, learned of this aim and asked for an unofficial survey of the possibilities of such a procedure. Nearly a year later, specifically in February of 1942, he informed the P A C O that the Kellogg Foundation had determined to finance such a venture as part of its international policy, and proposed that the PACO arrange annually for twenty-five Latin-American graduate students in ophthalmology to come to the United States for study. Each fellow was

to receive one thousand dollars a year, which could be extended to two years in case of exceptional ability, in return for which he was to agree to return to his native country to practice and teach ophthalmology. There was no time limit set to the grant.

This marvellous generosity was the greatest boon that could possibly have been given to further the cause of interhemispheric ophthalmology and the officers of the PACO set themselves immediately to the enormous task that the administration of these scholarships involved. The first step naturally was to obtain the approval and cooperation of the Department of State. Without the wise guidance and the active cooperation of the Department, it would never have been possible to accomplish the task. Under the supervision of the Department, the general procedure that was to be followed was set up and has been elaborated to the present stage. Then it became necessary to enlist the services of the leading ophthalmological institutions of the United States, for it was to these that the holders of the Fellowships were to be sent for their training. The following clinics agreed to accept one or more Fellows:

Bellevue Hospital and College, New York City
Columbia University, Harkness Institute, New York City
Cook County Hospital, Chicago, Illinois
Illinois Eye and Ear Infirmary, Chicago, Illinois
Mayo Clinic, Department of Ophthalmology, Rochester, Minnesota
Massachusetts Eye and Ear Infirmary, Boston, Massachusetts
Memphis Eye, Ear, Nose and Throat Hospital, Memphis, Tennessee
New York Eye and Ear Infirmary, New York City
New York University, New York City
Northwestern University, Eye Department, Chicago, Illinois
Stanford Lane Hospital, Eye Department, San Francisco, California
Tulane University, Eye Department, New Orleans, Louisiana
University of California, San Francisco, California
University Hospitals, Eye Department, Ann Arbor, Michigan
University of Iowa, University Hospital, Eye Department, Iowa City, Iowa
Wilmer Institute of Johns Hopkins Hospitals, Baltimore, Maryland
Wills Eye Hospital, Philadelphia, Pennsylvania

In these clinics the Fellows were to be accepted as full-time residents with board and lodging if space was available. They were to be given the same didactic, laboratory, and clinical training as is given to our nationals. In their daily intimate mingling with the American residents of the clinic, much was to be gained by both parties. They were also expected to attend at least one national ophthalmological society meeting during the year.

Then came the task of publicizing these opportunities. By letter, by printed articles in the ophthalmological journals, and by notices in the general medical journals, the word was finally spread around so that within four months, applications began to be received. The first three applicants accepted were already in the United States on their own responsibility and most gratefully they received the extended Fellowships with assignment to a definite clinic. At this point it should be noted that these forerunners have already made a very marked and favorable impression, not alone by the character of their work, but equally well by their ability to fit into the foreign surroundings of an American ophthalmological clinic. Rapidly are they proving the wisdom of the Kellogg Foundation in establishing the Fellowships.

The requisites for appointment to a Fellowship are that the applicant be a graduate of a class A medical college and have served one year or more as general intern or an equivalent service. The age limit of thirty years is somewhat flexible. The ability to use the English language is of course an essential. Another essential is the ability of a candidate to fit into the American scheme of things which has to be estimated by a local committee. A letter of recommendation from the applicant's professor of ophthalmology must accompany the application together with the agreement of the candidate to return to his native country to practice and teach ophthalmology upon conclusion of his term of study.

The Department of State volunteered to pay the ordinary travel expenses of the Fellow between his native country and the clinic to which he was assigned as well as the return costs. Of course, the Department reserved the right to reject

any Fellow who for any reason might not be acceptable for admission to this country.

Then came the question as to how the Fellow was to be selected. At the suggestion of the Department of State, the P A C O was able to enlist the cooperation and aid of the Institute of International Education, a volunteer organization in New York City, under the able guidance of the erudite Dr. Stephen Duggan. This organization has played an important role in the selection and management of Fellowships in other fields of education and to that end has been instrumental in the establishment of local selection committees in the capitals and a number of other cities of the American Republics. Serving on those committees are local leaders in various educational fields as well as members of our embassy staffs, and for the specific selection for Kellogg Fellowships was added one ophthalmologist, appointed by the PACO Council member of that country. The method of appointment to Fellowship is now as follows: The candidate sends a formal application accompanied by a letter of recommendation from his professor of ophthalmology to the local Committee on Selection of the Institute of International Education. A personal interview follows. The decision of that committee as to the suitability of the candidate, together with all papers, is sent to the secretary of the PACO (Dr. Alvaro for all countries south of Panama, and Dr. Berens for the remainder), who in turn forwards all papers with his recommendation to the Institute. The Department of State is consulted as to eligibility of the candidate for admission. The recommendation of the Department of State is added and all papers are then submitted to the Kellogg Foundation which in consultation with the president of the PACO makes the final decision. If favorable, the accepted Fellow is notified by the PACO and by the Department of State and is given specific instructions as to where and when to report. This procedure may sound somewhat complicated, but is absolutely necessary because so many agencies are of a necessity involved.

Thus far 12 Fellowships have been granted and the recipients are either in this country or are en route. Inasmuch as travel is so difficult during this wartime, the allocation according to population has not been followed and an unduly large number have been allotted to nearby countries from which travel is less complicated. For normal times, the allotment of Fellowships to the various countries is as follows:

Chile	2	Panama		
Cuba	2	Honduras		
Uruguay	1	Nicaragua		
Paraguay	1	El Salvador		
Peru	1	Costa Rica	2	
Bolivia	1	Guatemala		
Ecuador	1	Haiti		
Colombia	1	Dominican Republic		
Venezuela	1	Puerto Rico		
		Mexico	2	
		Argentina	4	
		Brazil	6	
		Total	25	

(Continued on Page 44)

THE PAN-AMERICAN CONGRESS OF OPHTHALMOLOGY

To INSURE the future of the Pan-American Congress of Ophthalmology a full membership is essential. If you are not already a member you are urged to complete this blank and send it with your remittance of $5.00 to cover membership fee, dues for 1944, and one year's subscription to OPHTHALMOLOGIA IBERO-AMERICANA.

Please remit to **DR. CONRAD BERENS,** ***Secretary-Treasurer***
477 First Avenue, New York, N. Y.

Name.....

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But the PACO is not content alone to bring Latin-Americans to the United States for graduate study. Such a course reflects a chauvinistic egotism that does not and should not exist. A program is now in course of development whereby clinics in Latin-America that are equipped to carry out an adequate graduate teaching program will be asked to accept Fellows from other Latin-American countries and eventually Fellows from the United States. Naturally there are fewer such clinics than in this country but it is believed that a true international rapprochment can gradually be attained.

The second meeting of the PACO was scheduled to be held in Montevideo, Uruguay, November 4-9, 1943. Under the able leadership of Professor Vasquez-Barriere, an active local committee was engaged in completing necessary arrangements. An attractive scientific program was set up by a committee consisting of Professor Vasquez-Barriere of Montevideo, Professor Charlin of Santiago, Chile, Dr. Alvaro of Sao Paulo, Brazil, and Doctor Berens of New York. It was as follows:

1st Day—Thursday, November 4: Inaugural Session.

2nd Day—Friday, November 5: Social Ophthalmology.

Morning: **Prevention of Blindness in the Americas.** Prepared by the Committee on The Prevention of Blindness of the Pan-American Congress of Ophthalmology; Chairman, Dr. Francisco Belgeri of Buenos Aires, Argentina.

Afternoon: **The Status of Trachoma in the Americas.** Prepared by the Committee on Trachoma of the Pan-American Congress of Ophthalmology; Chairman, Professor Ivo Correa Meyer of Porto Alegre, Brazil.

3rd Day—Saturday, November 6: Research in Ophthalmology. (Eight studies to be selected by the committee.)

4th Day—Sunday, November 7: Excursions and Festivities.

5th Day—Monday, November 8: Medical Ophthalmology.

Glaucoma

1. The Preglaucomatous State—Its Diagnosis and Treatment.
2. New Ideas on Glaucoma Derived from Gonioscopy.
3. Estimation and Mechanism of the Destructive Effects of Ocular Hypertension.
4. Surgical Intervention in Glaucoma—How Far Can Medical Treatment Be Continued.

But conditions entailed by the war will probably necessitate the postponement of the Congress for a year. At present a mail vote of the Council is in progress and all indications point to the unanimous decision that a much more satisfactory meeting can be held in 1944 than in 1943.

And such is the history of the Pan-American Congress of Ophthalmology from the period of its inception to this date of May 1, 1943.

HARRY S. GRADLE

Symposium . . . Seeing, Hearing, Thinking and Doing —

A Problem of American Youth

CHARACTER AND THE SPECIAL SENSES

JOSEPH E. RAYCROFT, M.D.

PRINCETON, NEW JERSEY

BY INVITATION

INTRODUCTION

FEW SPECIALISTS in any branch of medicine or surgery have made greater contributions to the everyday comfort and efficiency of modern man than have the members of the groups that compose this Academy in the promotion of the function and integrity of the special sense organs.

You have utilized all modern scientific discoveries that contribute to the diagnosis and measurement of defects of the eye or ear or other special sense organs; and in addition, you have utilized any new method or material that gave promise of aid in the correction of or compensation for any defects or handicaps that might be present.

The realization of these facts would tend, quite understandably I am sure, to arouse in a man whose experience has been largely in other fields of medicine and who has been invited to address this group the impulse, if he were *wise*, to select a topic as far removed as possible from any of the specialties here represented.

However, the die is cast and so I shall spend a few minutes discussing a seldom mentioned but important element in the field of your activities — the influence of the Special Senses on the Character and Achievements of the individual.

Presented at the Forty-Eighth Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 10-13, 1943, Chicago, Illinois.

DEFINITIONS OF TERMS

In the first place, it will be wise to make sure that we all have the same general understanding of the definitions that I have in mind when I refer to the term "character" used in the title of this paper. The reason for this precaution will be apparent when I call your attention to the fact that Mr. Webster, in his famous "Unabridged," found it desirable to subdivide his definition of *character* into *twelve* separate sections, plus a few subsections, in order to avoid any misunderstanding of his concept.

I have selected for our purpose *Section 5*, somewhat condensed, as follows: "Character is — the sum of the qualities or features of an individual — or the aggregate of mental or moral qualities — or the stamp impressed by nature, education or habit." Or we may interpret the "*Personality* of the *Individual*" according to the concept of Doctor Plant that: "Personality is the Person Behaving."[7] After all, this latter is a pretty reliable criterion and one that we shall find most dependable in our study.

TYPES OF PERSONALITY

We are all familiar with the wide differences — the pleasant and the unpleasant, the stable and the unstable, the bright and the dull, and so on to the end

of the list — in the personalities of those with whom we come in contact.

Most of us are satisfied, if we consider the matter at all, to ascribe these varying characteristics to vague factors of heredity or environment which are difficult or impossible to modify in a given individual, or to adopt some obvious explanation that is based on a history of accident or association.

Now it is against this background that I want to discuss certain facts and concepts, connected with the field of your interest, that illustrate the very important role played by certain of the special senses — functioning well or ill — in molding personality, influencing the development of character and affecting the quality of accomplishment.

INFLUENCE OF SENSORY DEFECTS ON HUMAN EFFICIENCY

This concept may be clarified somewhat by a few specific groups of instances illustrating the association of defective physical conditions — possibly unrecognized — and unsatisfactory accomplishment or psychological "twist," which represent either a valid *cause-effect* relationship or a very striking *set of coincidences*.

Further, for the sake of brevity and because there is a wealth of material based on reliable observations in the fields of *seeing* and *hearing*, we will limit oursleves for the moment to eyes and ears.

The knowledge of the relationship between grade of performance and efficiency of sight or hearing is so common that one needs only to note the results of a few surveys of typical situations to serve as illustrations.

We will assume that during the first six years of life the eyes have become coordinated, accurate function with binocular fixation has been achieved and the development of the eyes and their functions are relatively normal.

Following this period, the influences of school life operate to bring about or disclose sight deficiencies, which in a striking precentage of the school population are associated with unsatisfactory school performance, as shown by the results of the following surveys.[4]

One survey shows that about 66 per cent of the children in school make normal progress in their studies. Of these, only 15 per cent have eye deficiencies; while of the remaining 34 per cent who fail to make normal progress, examinations showed that about 85 per cent of them suffered from defective vision.

In another survey of grade schools the pupils were divided into three groups — superior, normal and retarded — according to progress in school work. The percentage of defective eyes in each group was as follows:

In the superior group.....14 per cent
In the normal group.....25 per cent
In the retarded group.....32 per cent

SOME CONTRIBUTING FACTORS

We must not overlook the further fact that the quality and amount of light available in school rooms have a positive and immediate effect upon the nerve-muscle tension as well as the working capacity of the student, even when his vision is normal.[5] This effect has been demonstrated by accurate measurements of changes in nerve-muscle tensions of readers who were *unaware* of the fact that their reactions were being measured and recorded. These observations demonstrated dramatically the *relaxation* of tension as the quality and amount of light available for the reader was *improved*, as contrasted with an *increase* in the recordings of nervous tension when the quality and amount of light were *reduced*.

These observations indicate that the effect, on even a normal visioned person, of long continued use of the eyes for close work, with insufficient light,

would develop in the reader a condition analogous to that mentioned by Aldous Huxley, who refers to "occasions when he was overcome by that sense of complete physical and mental exhaustion that only eye strain can produce."[2]

PSYCHOLOGICAL EFFECTS OF SEEING OR HEARING DEFICIENCIES

The records quoted so far refer mainly to the relationship between deficiencies in seeing and the *quality* and *amount* of work accomplished. However, we must not overlook the importance of the *psychological* effects of these conditions, which frequently pass unrecognized; such as, discouragement, nervous tension, feelings of inferiority and other undesirable psychological conditions.

When we turn our attention to the effects of deficiencies in hearing, we are impressed with the fact that while they also serve as serious handicaps to the learning process, the psychological effects are even more marked than those that accompany deficiencies in seeing.

We are fortunate in having testimony available on this point from a most intelligent and reliable source — Helen Keller — which will be most useful in our consideration of the effects of deficiencies in this particular field. Miss Keller stated to Dr. Joseph C. Solomon, a psychiatrist at Mt. Zion Hospital in San Francisco: "I consider my deafness to have been a much more serious handicap to me than my blindness."

Doctor Solomon,[9] in discussing this statement and the effects on personality of hearing defects, says in effect: "It is difficult for anyone in possession of all his faculties to evaluate the relative importance of vision and hearing. It is our natural tendency to feel more sympathy for the blind than the deaf, whether the condition is congenital or acquired.

Careful studies based on critical observations indicate that persons, particularly children, who are deaf or hard of hearing are much more subject to personality changes than are those who may suffer a corresponding degree of deficiency in the field of sight.

"One of the most important factors in this difference is the attitude of the parents. It frequently happens that parents do not recognize the hearing handicap sufficiently early to avoid going through a period when they look upon the child as stupid or negativistic.

"Even when they fully recognize the handicap they frequently and unconsciously take a hostile or unsympathetic attitude toward the child. The result is that the child feels unwanted and unloved."

A deaf person is apt to be suspicious of conversations that he hears only imperfectly, and frequently this impression tends to the development of a paranoid reaction. This may be aggravated by teasing playmates, whereas children almost never tease a blind child.

It is not surprising then to learn that it is estimated that six times as many deaf persons as hearing persons, in proportion to their relative numbers in the population, develop psychoses that require hospitalization.

Doctor Solomon emphasizes the importance of early preventive treatment to protect the hearing, and in cases where these efforts fail wholly or in part the emphasis should be placed upon early diagnosis of the extent of deficiency, followed by training in the use of hearing aids such as lip reading, etc., as a means of preventing or modifying the developments of personality problems.

INFLUENCE OF THESE FACTORS ON GENERAL BIOLOGICAL DEVELOPMENT

We have given some consideration to the influences on *Personality* and *Performance* of certain deficiencies in the functions of the special senses. Now I wish to call attention as briefly as pos-

sible to the influence of these *deficiencies* in another and most important field, namely, the *Biological Development* of the *Individual*.[3]

This effect depends upon what Professor Herbert S. Jennings called the rule of "Attention," which he describes as one of the three important "rules of development!"

1. The rule of the gradual and spontaneous development of the powers.
2. The interdependence of the physical and the mental, and
3. The rule of "attention" in physiology and development.

Rule 3 represents a principle that is of the utmost significance in its bearing on our problem.

The principle is this: "Throughout development and all activity, both bodily and mental, there holds a rule which is comparable to the ordinary rule of attention in the activities of the mind."

This expression is not precise, but is in the nature of an analogy which will serve as a guide in complex situations — "Only one thing can be attended to at once."

This means that: "When the energy and the attention of the organism are thoroughly absorbed in one activity, physical or mental, other activities do not prosper; for instance, when the organism is taken up with an intense emotion, particularly if painful, the digestive process stops. Intense physical pain or intense mental application have a similar effect." Any continuous pain, such as from diseased teeth or overstrained eyes, tends to retard other physical and mental processes.

NEW DEMANDS ON SPECIAL SENSES

Our consideration of the vital importance in everyday life of normal and well functioning eyes and ears would be seriously incomplete if we failed to recognize and appreciate the significance of the revolutionary changes that have taken place in our pattern of life.

These changes need only to be mentioned to make us realize that in their train have come previously unheard of demands and responsibilities on the special senses.

The barest outline of what has taken place during the past 100 years, with a great increase in tempo during the past 20-25 years, may illustrate this point.

The first period — approximately fifty years: Simpler life, less mechanical, smaller proportion of time at *books* and *other close work*. Seeing, hearing and body balance developed by hunting, fishing and other outdoor activities in the form of both work and play.

The second period — up to World War I: Larger percentage of time in school at close work, frequently inadequate light, more reading or other close work at home. Less demand on eyes and ears, or upon semicircular canals for orientation and balance.

The present period, since the last war: Still further emphasis on sedentary education, together with increasing *demands* on all *special senses* and dependence on their messages — motoring at high speed, safety depending upon keenness of sight, wide field, speed of adjustment, stereoscopic vision, judgment of size, speed and direction, and proper responses to meet rapidly changing situations.

Look at our record of motor accidents due to defects in these adjustments — aided and abetted by atrocious road manners. If you don't see the right hand side clearly then "straddle the white line."

Consider the *demands of aviation* on eyes, ears, semicircular canals and vasomotor system.[1] There has been nothing like it in any previous existence! It is a wholly new chapter in human experience and can be barely mentioned here.

SUMMARY AND CONCLUSIONS

A comparison of the reasons for rejection of draftees in the medical examinations of the draft boards in 1917-18 and in this war shows a striking similarity in groups of causes responsible; during the present emergency the rejections for all causes are approximately 50 per cent as compared with 30 per cent in World War I.

These results 25 years ago jarred our complacency — we made speeches, we wrote papers, we even passed laws providing, among other measures, for the detection and treatment of bodily weaknesses and the deficiencies of the special senses as a part of the responsibilities of the public school!

How efficiently was this program administered? Children born between 1912 and 1919 were among the first to have the opportunity to profit from lessons learned from the examinations of World War I, as these lessons found expression in the systems of education and medical supervision in the public schools. The results were disappointing.

The number and percentage of rejections due particularly to deficiencies in the special sense organs and to mental and emotional weaknesses that have been disclosed in the current draft examinations indicate that we have failed pretty completely to profit by our experiences in these particular fields during World War I and to live up to our brave resolutions after it came to an end.

So when we come to study the school record of those rejected because of deficiencies of sight or hearing in the current draft examinations, we learn that *50 per cent gave evidence of these defects when they were in school.*[8]

The fact that these crippling conditions were recognized years previous and were not given the benefit of corrective measures, but were in fact practically ignored, is disturbing, not to say humiliating.

Further, we should not lose sight of the significant fact that defects of the special sense organs that render one ineligible for military service also operate as more or less serious handicaps to the attainment of efficiency and satisfaction in civilian activities.

Is there any solution to this problem of the special senses? An unqualified affirmative doubtless is unwarranted. On the other hand, there is sufficient evidence at hand to warrant the expectation of achieving a large measure of success in preventing or correcting undesirable intellectual and emotional consequences due to defects of the special senses, providing adequate use is made of knowledge generally available or easily obtained.

THE SCHOOL PROGRAM

In the first place, the necessity must be recognized of dealing with the child of school age as an individual with individual characteristics, problems and needs which call for a certain minimum of individual help and adjustment.

This means that the scope and objectives of school medical examinations and supervision must be more clearly formulated and administered on a more realistic and practical basis.

It is no longer sufficient to concentrate on the discovery and control of acute infections, important as that function is. Nor does the addition of procedures to discover tuberculosis or to protect against certain infectious diseases by specific vaccinations meet the needs disclosed by these surveys of manpower and the reasons for disqualifications.

Any proposals of the type referred to will inevitably give rise to important questions:

1. Who is to have the responsibility for organizing such a program of supervision?

2. By whom shall these important medical examinations be made?
3. How will the expense involved in the examinations and necessary treatment be met?

These questions and others of the same type will not be found *unanswerable* if the vital importance of the job is realized by the public and emphasis is placed upon high professional standards and efficient service.

It must be recognized at once that any program along the general lines suggested here will have little or no chance of being formulated and put into practice unless there is developed a widespread conviction of the profound importance of the functions of the special senses — hearing, seeing, smelling, touching, etc. — as points of *contact* and *avenues* of *relation* with the *environment*.

This academic recognition must be supplemented by an active campaign to bring home to physicians, parents and teachers the importance of promoting and protecting in every way possible the normal function of these sense organs and their appendages. In no other way can we *insure* to the *individual* child the ability to make prompt, reliable and consistent recognitions and evaluations of his contacts with the outside world.

The importance of early, skilled and constant attention to the normal development and function of these "Gateways" cannot be overemphasized and has long been recognized. The old Elizabethan Schoolmaster, Richard Mulcaster, wrote nearly 400 years ago: "These three things — perception, memory and judgment — are the basis of education." Also, Mach[6] stated much more recently: "The facts furnished by the senses are alike the starting point and the goal of all mental adaptations."

Our interest in the war effort naturally and properly emphasizes our concern when we realize that defects of eyes and ears are responsible for about 20 per cent of all disqualifications — in the age group 21-36 — for any type of active military service.

We must not, however, let our disappointment at the loss in military strength make us unmindful of the much greater number and importance of such defects among men, women and children in civilian life.

It is very difficult to evaluate the losses in happiness and efficiency suffered by individuals and by society as a whole.

Take for example the increase in expense and the decrease in efficiency of our educational program, caused by eye and ear deficiencies among a considerable proportion of the students.

The whole student group is perceptibly retarded — the normal student loses interest when the pace is too slow, the efficiency of the instructional staff is reduced, while increases in expense of plant and administration bulk large in the budget.

AN OPPORTUNITY FOR PUBLIC SERVICE

No other professional group is as well qualified as the medical men to furnish leadership in this field to parents, teachers, school authorities and the public, by reason of their training in the developmental factors involved in this problem of youth.

This is a many-sided problem, the solution of which will require taking into account every aspect, mental, physical and educational, of the development of the human individual.

There are few fields of medicine and education where there is such an opportunity for public service, and where success in bringing about improvement in the education and protection of the special senses will be followed by such satisfactory and valuable results to both the individual and society.

And finally, Mr. President, if you and your associates can establish an adequate program of supervision and protection of the special senses from childhood on, you will merit the depth of appreciation expressed by Winston Churchill when he said, "Never have so many owed so much to so few."

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Symposium . . . Seeing, Hearing, Thinking and Doing —

A Problem of American Youth

THE OPHTHALMOLOGIST'S ROLE IN THE HEALTH PROBLEMS OF AMERICAN YOUTH

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THE RECENT national statistics on the physical fitness of the youth of America reveal what one would believe to be serious ocular defects. The high totals would lead one to believe that the care of the eyes of these individuals has been deficient. A more careful study of these figures reveals that there has been little or no change in ocular problems during the past 30 or 40 years. The figures do not indicate a marked degeneration of the eyes of our people, but they do show for the first time that by putting the micrometer to a natural, physical, anatomical structure nature builds to a pattern but not necessarily to an optically, mechanically perfect one. The tolerances according to engineering principles are great; however, in the eyes the tolerances have been narrowed to a point beyond nature's ability to respond. As future detailed figures are presented and the various etiological factors are revealed, we will be better fitted to discuss the problem from a scientific viewpoint. Even though the figures do not show a definite increase in ocular pathology, they do present a definite challenge to ophthalmologists to make an attempt to diminish as much as possible the extremely high percentage of rejections of men for military service due to ocular disease.

Figures presented by Dr. Leonard G. Rowntree in the September 25, 1943, issue of the Journal of the American Medical Association show that rejections because of ocular defects head the list in the group of individuals of the white race, and ocular defects in the group of colored individuals are seventh in the list.

What is the reason for such a large number of youths with ocular deficiencies? Is it due to the lack of medical care, faulty parenthood or faulty nutrition, or is it the normal percentage of natural defects for so large a group of individuals? It is intimated that there is a definite lack of medical care.

In order to keep the records straight, when the Ophthalmic Board was formed in 1916, it was the same as giving the public a gilt edged certificate that designated which men had the approval of the Board and were guaranteed as specialists in their field. Subsequent to this the Academy, in a farsighted procedure, began its instructional courses which was a purely educational effort. It not only tends to but actually does keep all its members abreast of the new medical and surgical procedures and discoveries, and it also helps them in their review of

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special subjects. These specialists in ophthalmology and otolaryngology are well trained and have been kept well posted. The failure is therefore not the fault of these specialists as individuals; however, there might be some criticism as to their available numbers. The caliber of these men could not be increased by government control, nor could any system of government guarantee better training or better follow-up instruction. There might be one other point of criticism: Due to their reticence these specialists may have been excluded from taking part in national problems which they and they alone would be best suited to handle. It is not due to their lack of interest in national affairs or educational matters, but rather to the fact that members of the medical profession as a rule do not make any suggestions in national and educational matters without first being asked for them.

That definite ocular problems exist during youth cannot be questioned. The first and most important factor concerning sight is that of heredity. In a country where we have not only a democracy but practice democratic principles, youth decides for itself whom it will marry and who will be the father or mother of its children. This process of natural selection is ideal, democratically speaking, but from the point of view of genesis this is not the most expedient method.

Let us consider those countries in which myopia is an outstanding physical defect. Would it not be better for them to have, as we do here, the large number of racial strains that do not show myopic tendencies? Thus, myopia is kept at a constant level in this country. Other dominant physical characteristics would likewise tend to be held in a minority where there is so much intermarriage of people with different hereditary strains. In this country with its multitudinous races, heredity may play a part. Some people are mentally superior and others are physically superior. In our democracy the only control we have over an individual is by education. By this means knowledge will supplement emotion in the matter of marriage. It would therefore seem impossible for hereditary factors to be regulated under our present system except by education.

A great deal can be done to control the second major factor, which is the so-called neuromuscular or psychosomatic factor. This concerns physical and psychological hereditary features. Without question much can be done in any group to improve the character and personality of the individuals. The numerous anatomical, physical and psychological defects can generally be corrected or altered to the benefit of the individual.

We ophthalmologists have an important and large field. The direction of the early treatment of those who have defective vision is necessary because of the far reaching effect on the individual. Some of the cases require early surgical interference, such as the removal of cataracts, in order to give the child the earliest opportunity for education; other cases require treatment and thorough investigation as to the patient's psychological state. This is especially true in reference to those patients with muscle imbalance. There are many physical and psychological inequalities in nature, and it is up to the medical profession to improve the status of any individual, when it is possible to do so, by medical procedures. There is a possibility here for criticism in the handling of patients with muscle imbalance. In many instances, by careful training the child will receive a second useful eye.

Several authors on this scientific program are taking into consideration office therapy in cases of strabismus. I do not believe that the problem ever will be

solved satisfactorily until many children, mainly the intractable cases and those with difficult problems which are associated with psychological trends, are treated as institutional cases. Much time can be expended in trying to raise the level of a nit-wit to a half-wit if the basic mental level of the patient is not known. This is not discovered by office observation, but by careful, well-grounded, prepared tests. It is my belief that in this type of case our tendency has been to allow the indifference of the parent to influence our methods of treatment much to the detriment of the patient. We must also educate the parent if we would alter the situation materially.

At this time another serious problem should be considered, and that concerns examining the child who is preparing to enter school. The early education of the child necessitates practically all visual functions, and unless the child's vision is adequate he may develop serious psychological trends which will interfere with his entire future education and affect his character. It has been my contention that the ophthalmologist should take part in directing the pre-school child's early education along visual lines, that every child is entitled to a complete eye examination and that his visual quotient should be a part of the school authorities' record by the time the child reaches the reading age. There are so many children now wearing glasses that those who do wear them no longer are subjects of ridicule for their playmates.

There is a far more important group which consists of those who have a manifest squint. Without question, the most out-spoken period of an individual's life is when he is a child, and he definitely makes the most of his comrade's physical inferiorities for his own pleasure. The cross-eyed youngster is subject to constant and disagreeable remarks, as well as being made aware of his own appearance. This frequently leads to a very serious inferiority complex which is difficult to overcome and may cause various types of juvenile delinquency and difficulties with school authorities.

Our mere saying so does not alter the psychology of children, and I believe even though the child has a central field defect and his vision cannot be improved, an effort should be made to correct the squint in a child before he enters school. The vision of a child during early school life is important, and we ophthalmologists have not taken as active a part either in regulating education or in guiding school authorities. There is a possibility for much research and investigation to aid them in their decisions. This might well be a voluntary contribution. The age at which children start to read and use their psychosomatic faculties differs and is not related to their physical age, but primarily to their mental age. The psychological upheaval may be terrific because of the child's unreadiness to begin his education. To be sure, we have never been asked for aid in this field, but I believe this organization might appoint a committee to make worthwhile suggestions to school authorities, and not leave the decision to people who are not qualified to make ocular examinations and who are not always thinking of the best interests of the individual.

The field of education which is third in importance depends on the psychosomatic state of the individual, namely, his readiness for education. Although we Americans believe that we are the best educated group of people in the world, the fact remains that this is not entirely true when considering the country as a whole.

Education should be regarded in its broadest sense, rather than by the number of years spent in school. Frequently, education is forced without considera-

tion for the aptitude or desires of the individual. Large communities do a better job of directing students according to their aptitudes than do smaller communities; most are still in a quandary as to how to screen children with visual defects, when a child should start to read, and most important, what factors are most necessary for good reading. We all know by experience that when there are a large number of treatments for a specific disease the right treatment has not yet been found. School systems are more variable than are the numerous types of treatment advocated for trachoma — the sulpha drug of education has not yet entered the field.

For example, the advice to the myope is often that of restraint, invalidity and discouragement, whereas the situation might well be approached by selecting new fields for the youth and encouraging him, and probably rarely ever putting him to total rest with loss of years of education. The replacement therapy must be undertaken at once, for falling behind in class and mental indolence are extremely degrading for youth.

There is one definite error in visual education, and in view of the fact that most all early education is promoted along visual lines, this error is serious, because insufficient time is devoted to teaching the youth how to see and what to look for. Unless his basic mosaics, as described by the Dartmouth group, are fairly distinct he would be an indifferent and inaccurate observer all his life. Time spent on each individual at this time is worth years of remedial reading and training later. The original effort should be made by individual instruction, to assure once and for all that the child recognizes what he sees. The ability to improve vision by educating the child to observe has promoted a lucrative racket for some of the off-shoots of our profession. It has given several pseudo-scientists the opportunity to write broadly and unwisely on the subject, and has necessitated all of us to be confronted with numerous, silly questions.

The fourth major factor in the problem of our youth is that of experience. By the time the individual has had proper education he will accept the experience of others, and will not try to solve all the problems by the trial and error method. Experience is probably the one big factor in life that we as medical practitioners can pass on — experience that is based on excellent observation and proper education. The youth of today has not been neglected by the profession but never in the history of mankind have the requirements been so high, and the demand for physically perfect specimens been so great. Nature does not always create physically perfect specimens, but this is what is expected in those candidates for aviation, the navy and marine corps, and this lack of physical perfection is the reason for the major number of rejections. Youth must depend on our experience for information concerning its physical, visual and psychological fitness; each is a major factor that can only be judged by people best qualified to study them. The future of our country depends on youth. His mental state and his reactions depend on our leadership. We cannot decide for him but we can guide him.

Two major factors obstruct the position of the ophthalmologist in the guidance of youth: (1) The insufficient number of oculists to cover the field of ocular examinations thoroughly as it should be covered, and (2) the sentiment that early visual education and training is not a part of their work.

In answer to this, all one can say is that, first, more men should be trained. Only by education and training will this ever be accomplished. By lowering the standards or by accepting poorly educated or incompletely trained men into

the fold will not solve the problem. Neither can we fill our ranks to overflowing with much "technical" addition without interfering with the progress of ophthalmology. Second, vision in its entirety is our problem, and it is our duty by virtue of our training to take an active part in the care of the eyes of individuals who are in the first two decades of life.

Symposium . . . Seeing, Hearing, Thinking and Doing —

A Problem of American Youth

THE OTOLARYNGOLOGIST'S ROLE IN THE HEALTH PROBLEMS OF AMERICAN YOUTH

A. C. Furstenberg, M.D.

ANN ARBOR, MICHIGAN

This is an age-old problem; one that has been with us since time immemorial and it should rightfully be emphasized during a national emergency. Doctor Raycroft has given us a vivid description of the tragedies of impaired hearing. He has approached the subject in the light of psychological factors which dramatically express the physical, moral and sociological influences of serious auditory impairment. His speech pulsates with human understanding and sympathy for patients thus handicapped, and his timely presentation will add an impetus to the powerful forces that are moving toward the satisfactory solution of this important public health problem.

I need not take your time to point out the old story of the incidence of hearing impairment; that in the United States between six and seven per cent of our population are afflicted and that by comparison to little Sweden, where annual testing of all school children and adequate follow-up methods have been conducted for the past 25 years, less than one per cent of the population suffer from impaired hearing. The scope of the problem, its recognition, the value of enlightened legislation — all have been exhaustively treated by numerous welfare agencies and by the medical profession, to the end that reiteration of this data has no place of importance in this meeting.

The thing we need to emphasize now is a plea for concerted action. Now is a most appropriate time for the unification of all forces, which have long labored on this problem, into an organization which will function for the nation as a whole, with clarity of objectives and precision of action.

It is most gratifying to observe that such forces are being crystallized into a program by the American Academy of Ophthalmology and Otolaryngology and the National Research Council which gives promise of effectiveness such as we have hoped for since otolaryngology became a specialized field of medicine. The joint collaboration of this dynamic Academy and the National Research Council should lead to a plan of national scope which will prevent and ameliorate hearing defects and rehabilitate those persons who have lost serviceable hearing.

The objectives of such a program are clear to all of us, but the means of attaining them will require unprecedented effort on the part of the otolaryngological profession, the ancillary service in the health sciences and our na-

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University of Michigan Medical School, Ann Arbor.

tional agencies. It is our solemn obligation, first, to identify and recognize those individuals who are hard of hearing; second, to provide adequate therapeutic measures for those who show promise of a favorable prognosis; third, to rehabilitate those individuals who require special education by remedial instruction in speech reading, voice correction and vocational guidance.

As I see it, most facilities are now available for those purposes with the possible exception of an adequate supply of teachers of speech reading. The present number of competent instructors of visual speech is entirely inadequate to meet the need as estimated from general statistics of the acoustically handicapped patients and the special conditions developing in a country at war. The necessity of academic programs for the education of speech reading instructors is at this very moment conspicuously in evidence in all our institutions for human adjustment. Groups of young people on an advanced professional level must be given adequate training. The importance of speech reading in the rehabilitation of the acoustically handicapped person cannot be emphasized too strongly. Its acquisition speaks for the return of normal social relationships through seeing speech which is neither not heard at all or is heard inaccurately and thus insures normal psychological states so essential to the patient's success and happiness. We are fully aware that science has brought to the person with auditory impairment the aid of modern electrical hearing instruments, yet otologists and laity alike bear witness to the fact that these aids often render service which is either incomplete or not effective at all when progressive deafness finally reaches a profound state. When the hearing aid is of service to the patient there is nothing in the use of the instrument which militates against the usefulness of speech reading. The two are frequently supplementary, each augmenting the service of the other. The hearing instrument, however, may cease to be an aid when critical levels of hearing loss are attained, while the value of speech reading never depreciates except in cases of temporary auditory impairment.

Let us, therefore, mobilize all our resources in the interest of the welfare of the many thousands of persons now handicapped by hearing defects. The facilities for this worthy program and the impetus for their prompt utilization are now made possible by a public spirited profession and receptive federal agencies. It is our solemn obligation in the face of this emergency and during the period of reconstruction to exert every force and to employ all available means to promote normal human adjustments. The problem of the acoustically handicapped individual is but one of our great welfare obligations in which we as a strong organization of otolaryngologists can render a noteworthy service.

A CLEAR CORNEAL IMPLANT ACQUIRES DYSTROPHY FROM ITS HOST

ROBERT VON DER HEYDT, M.D.
CHICAGO, ILLINOIS

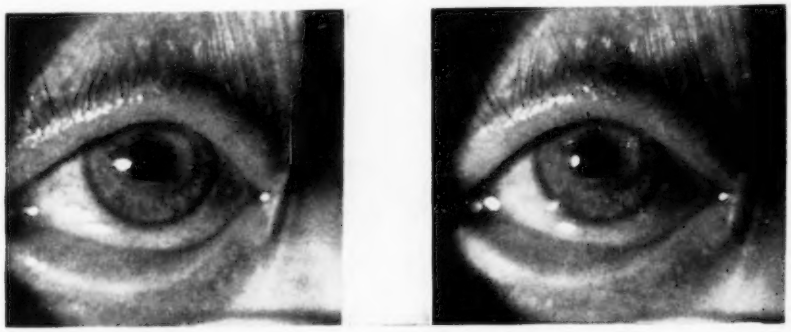
TO ASSURE a permanent transparency of clear implanted corneal tissue, the window must be imbedded within a partly clear stromal area of the host's cornea. Transplants placed within leucomata soon become clouded.

Twenty years ago Salzer[4] asserted that corneal cell activity of the host incites a response within the implant. This would induce a slow metamorphosis and the gradual replacement of the implant by regenerated tissue (keratoblasts). The infiltration of the implant takes place with a very mild reaction if the tissues of the host are relatively similar and therefore receptive to the individual chemism of the implanted tissue.

Castroviejo[1] after microscopic examination of transplanted corneal tissue came to the conclusion that this replacement by tissue of the host does not take place.

Friede[3] also does not believe that new clear tissue within the implant can be derived from the host. He implies that corneal corpuscular division may take place. The development of scar tissue within transplants proves to him that connective tissue elements may form independently of the host.

Eyes which have lost useful vision because of a corneal dystrophy are among the best suited for successful corneal replacements. These eyes are usually normal in other respects. In dystrophy the process involves only part of the clear stromal tissue. This factor enhances the viability of the implant.



Clear corneal implant acquires dystrophy from its host.

Friede,[3] Franceschetti[2] and Castroviejo[1] pioneered in placing transplants into corneae affected by hereditary dystrophies.

In 1938, I[5] was invited to contribute an article to the Vogt Festschrift. My subject was "Hornhautdystrophie und Plastik." I emphasized the suitability of corneal transplants in the treatment of

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corneal dystrophies and included kodachrome photographs taken before and after the corneal transplants depicting the case I was about to present.

Authentic cases of invasion of clear corneal tissue implants by dystrophy from their hosts are not on record. Several instances are reported where a very early general clouding of the implants developed. Frieberg in a discussion made mention of a case in which a dystrophy had supposedly invaded by way of the peripheral edge of the implant. The type of dystrophy was not mentioned.

My patient, V. K., is now 52 years old. His dystrophy is of the granular disciform type — the most common of Buecklers Triad. He came to me over 20 years ago. I have had his slowly progressing dystrophy under constant slit-lamp observation, as well as photographic recording, during all this time.

His vision 20 years ago was 20/80 —. There occurred a slow gradual increase in the involvement of the central disc area of circumscribed granular bread-crumb like lesions imbedded within clear stroma. In 1933, 10 years later, because of the progression, his vision had been reduced to 10/200 —. Over six years ago I referred him to Dr. Ramon Castroviejo, who successfully transplanted a clear quadratic area of newborn cornea. His vision with correction was 20/50.

About four years ago I noticed a fine sub-surface salt granule-like change in the center of the implant. This was two years after the transplantation. After seeing this from time to time the thought occurred to me: What if this implant should acquire the dystrophy from the host? I consoled myself with the thought that, dystrophy being a hereditary characteristic, it cannot happen.

This minute change observed for years because of very slow progression has now assumed the definite characteristic picture of the host's dystrophy. There has been but little deterioration of the visual acuity because of the involvement of the implant.

Buecklers in 1938 obtained a specimen of this type of dystrophy for histopathological examination. There was an amorphous circumscribed deposition within all the stromal layers. The involved tissue particles stained red with eosin, olive-brown with Van Gieson, luminous red according to Passini and did not stain with Kresyl-violet. He considers the change to be keratohyalin in type, which originates in situ because of the degeneration of the stroma.

The regional involvement of this type of dystrophy, as seen in all incipient cases, is in the center as well as under Bowman's surface. This area is most remote from the entry of fluids derived from the host.

Franceschetti[2] did a series of transplants in cases of dystrophy of the cornea in 1935. Four years thereafter they were still clear. This to him proves Vogt's theory of the influence of genotypic changes within corneal tissue in contradiction to the neurotrophic theory of Groenouw.

It is possible that some day biological chemistry will establish as the basis for causes of certain hereditary changes the presence of specific substances circulating within the body.

In the case that I am presenting, the involvement within the clear implant is absolutely characteristic of the primary incipience of this type of dystrophy. Its very early manifestation is therefore also in the central zone and adjacent to the Bowman level. The margins of the implant are very clear. It reproduces the dystrophy in the implant in the same central location and apparently in the same mechanical manner in which it originally had involved the host. The

host's, may we say, change-inducing fluid, here at this area, most remote from the marginal entry, meets a less pervious barrier, a stasis, and the dystrophic change is the result.

This specific element or fluid, whatever its characteristic properties, initiates a definite type of dystrophic corneal change. It embodies the hereditary factor of necessity, common to all individuals who are so afflicted.

DISCUSSION

RAMON CASTROVIEJO, M.D., New York, N. Y: There is really nothing that I can say about Doctor Von der Heydt's paper except that he has with his characteristic thoroughness reported a case of corneal transplantation in which the graft has become affected with the same type of corneal dystrophy of the host. Dr. Von der Heydt has also expounded the theories which may explain the recurrence of the dystrophy into the graft.

I have had some other cases of corneal dystrophy, especially dystrophia adiposa in which the transplant has been invaded by the dystrophy of the host. In these cases in which the dystrophy is located on the superficial layers of the cornea a superficial keratectomy is to be preferred instead of a keratoplasty. If after the keratectomy, vision has not improved sufficiently, then the corneal transplantation is indicated.

I have not observed other invasions in transplants of the same type of dystrophy as it has been described today by Dr. Von der Heydt. I cannot advance any explanation or give any theory about the mechanism of the production of the dystrophy in the cornea that has been implanted. I can only congratulate Dr. Von der Heydt for his very thorough presentation and hope that some day biochemistry, as he has mentioned, will perhaps explain this very mysterious behavior of the corneal tissue.

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4. Salzer, Fritz: Uber Hornhautregeneration und ein neues Verfahren der Abrasio corneae, die Keratektomie, Ztschr. f. Augenh. 49:283, 1923.
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BARTHOLOMAEUS EUSTACHIUS:

His Contribution to the Anatomy of the Ear, Larynx and Bronchi

CHARLES J. IMPERATORI, M.D.
NEW YORK, NEW YORK

"ALMOST all scientists and technicians develop an interest in the history and progress of their science after they have acquired enough experience to realize that some of their predecessors have possessed many of the basic ideas and facts that have been studied and elaberated upon by succeeding generations."[16]

Many of the superb works of the early writers and masters in their particular field remain immured in archives or just as securely locked up in untranslated volumes of Latin script or foreign languages, just as the "Pirotechnia" of Biringuccio, published in 1540, was passed over most recently by the Hoovers in their translation of Agricola's De Re Metallica.

The accomplishment of a translation well done of any work is an indication that the translator's imagination has been so stimulated by the author that he can transpose the thoughts and ideas of the originator into another language, so that it is readable and understandable.

The magnificent anatomical drawings of Leonardo da Vinci were lost for over 400 years, and have only been known generally for the past 40 years. Similarly, the anatomical sketches and drawings of Eustachius were lost for over 140 years. The anatomical achievement of both Leonardo and Eustachius were much greater than the influence exerted.

FIG. 1
TITLE PAGE OF THE OPUSCULA ANATOMICA

"The anatomical works of Bartholomaeus Eustachius, the Sanseverino Physician and Philosopher.

The number of these works and their arguments are indicated on the reverse page.

With the Patronage of the Highest Pontiff, of the Illustrious Senate of Venice, of His Catholic and Imperial Majesty, King of France.

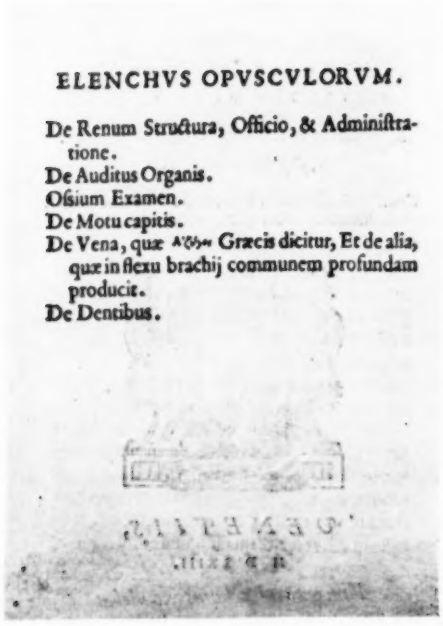
Venice, 1564.

Published by Vincentius Luchinus."

NOTE. — The signature of A. Flint, Jr., indicating ownership of this volume, is that of Austin Flint, Jr., Professor of Physiology at the Bellevue Hospital Medical College, circa 1880-1898. He was a collector of rare medical books. He was the son of Austin Flint, the great clinician and heart specialist of the post Civil War days, and who practiced in New York City and was Attending Physician to Bellevue Hospital. The present Austin Flint was also known as A. Flint, Jr., he being the son of the A. Flint, Jr., whose name is on the title page. He was Professor of Obstetrics at the Bellevue Hospital Medical College and Visiting Surgeon to Bellevue Hospital. He is now living in retirement at Millbrook, N. Y.

The writer had the privilege of having been a medical student under these two distinguished teachers.

Presented at the Forty-Eighth Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 10-13, 1943, Chicago, Illinois.



ELENCHVS OPVSCVLORVM.

De Renum Structura, Officio, & Administratione.
De Auditus Organis.
Ossium Examen.
De Motu capitis.
De Vena, quæ Ἄζυγος Græcis dicitur, Et de alia, quæ in flexu brachij communem profundam producit.
De Dentibus.

FIG. 2

REVERSE SIDE OF TITLE PAGE.
OPUSCULA ANATOMICA.
"AN INDEX OF THE WORKS

The Structure, Function and Care of the Kidneys.
The Organs of Hearing.
The Examination of Bones.
The Movements of the Head.
The Vein which the Greeks call . . ., and other things, which, when bending the arms, produce a common depression.
The Teeth."

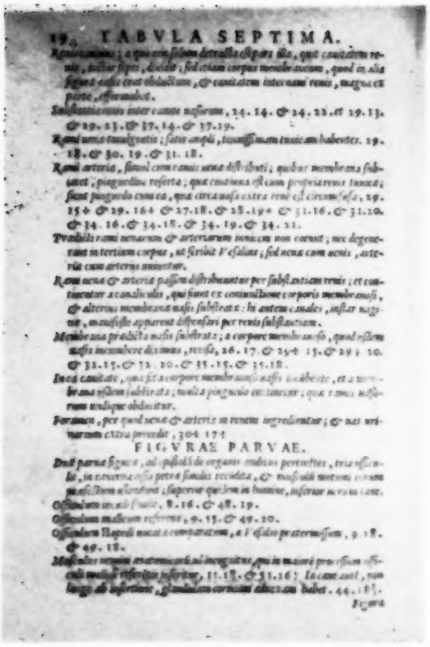
NOTE. — The outline of the frontisplate and some of the printing (Venetis MDLXIIII), etc., can be seen coming through the paper.

In the Opuscula Anatomica,[8] Fig. 1 and Fig. 2, which Eustachius published in 1564, there were but eight plates. These illustrations were of the kidney, Fig. 3, veins of abdomen, azygos vein, the heart and its valves and two small illustrations of the bones of the middle ear and tensor tympani muscle. Similar illustrations of the ossicles, the middle ear, the cochlea and the semicircular canals are shown in the Tabulae Anatomicae.[12] These are the copper plate engravings that were not published until 140 years after Eustachius' death. Other illustrations of the bones of the face, skull and bronchial tree and accompanying blood vessels are part of these Tabulae (Fig. 4). No illustrations nor descriptions of the accessory nasal sinuses are given. Examination of these illustrations of the frontal bones and the superior maxilla would convey the impression that they were not present. These Tabulae were drawn by Eustachius himself and his relative and assistant, Pier Pini of Urbino, and were engraved by de'Musi of Rome. This engraver was one who had done considerable work in architectural lines and this probably accounts[4] for the fact that the engravings are lifeless and stiff, and not at all adequate representations of living parts. However, as noted elsewhere, only eight illustrations, composing the total of 47, were published during the lifetime of Eustachius. The remaining 38 copper plates were intended for his great work, "De dissensionibus ac controversiis anatomicis." However, he died (1574) before his work was completed. In all, there are 47 (illustrations) engravings on the copper plates. One plate was engraved on the reverse side. These are the original plates published by Lancisi with his own commentaries, and copied and recopied by others.

Jo. Maria Lancisi was physician to Pope Clement XI and the volume "Tabulae Anatomicae" was dedicated to him.

There is also included a testimony of praise, a lauditory poem and several letters relating to the compiler and author, Eustachius. (Fig. 5.)

In that year, 1714, Lancisi had printed the 47 engravings and included a description of the anatomical parts and also indicated the structures after the manner of Eustachius; that is, each detail of the illustration could be located



19 TABVLA SEPTIMA.

Reuocamus ; a quo ex filum detrahit disparatio, qui cauitatem tenuis, stellæ figuræ, duxit ; sed etiam corpus membranaceum, quod in alia figura adhæret obducit, & cauitatem interamni tenuis, magnaex parte, efformauit.

Subsultantes ossis inter tenues nostrorum, 34. 14. & 34. 22. et 19. 13. & 19. 23. & 17. 14. & 37. 19.

Rami arteriæ tunalgratis ; satis amplæ, tamen minus tenuis habentes. 19. 12. & 30. 19. & 31. 12.

Rami arteriæ, simul cum ramis venæ distributi ; quibus membranæ subtenues, pinguedine refertæ ; quæ continua est cum propriæ tenuis tunicæ ; sunt pinguedo cum ea, quæ circumfusa extra tenuem est circumferentia, 19. 15 & 29. 16 & 27. 18. & 28. 19 & 31. 16. & 31. 20. & 34. 16. & 34. 18. & 34. 19. & 34. 21.

Probabilis rami venarum & arteriarum tenuium non earum ; nec degenerans in tertium corpus, ut scribit Vesalius ; sed venæ cum venis, arteriæ cum arteriis misceatur.

Ramuscula & arteriæ passim distribuuntur per subtilem tenuis ; et continuatur a canalibus, qui sunt ex continuatione corporis membranosi, & alterius membranæ nasi subtilis : In anteu canales, inflat nasum, manifeste apparent diffensi per tenuis subtilitatem.

Membrana probabilis nasis subtilis ; a corpore membranaceo nasi, quod ossium nasis incumbere diximus, tenuis, 26. 17. & 25. 15. & 29. 20. & 32. 15. & 32. 20. & 35. 15. & 35. 18.

Intra cauitate, quæ sit a corpore membranaceo nasi subtili, et a membrana ossium subtilitatis ; nulla pinguedo continetur ; quæ venas nostrorum undique obducitur.

Foramen, per quod venæ & arteriæ in tenuem ingrediuntur ; & nas irrigarum extra prouidet, 304 17.

FIGVRAE PARVAE.

Duæ parua figuræ, ad ipsius de organo auditus pertinentes, tres ossiculis, in cauerna ossis petrosi fundis recondita, & musculi motum eorum modificantem alentem, superest quidem in homine, inferius memoratur.

Ossiculum incudi simile, 9. 16. & 48. 19.

Ossiculum malleum referens, 9. 15. & 49. 20.

Ossiculum stapedi simile comparatum, et stapedis præterea simile, 9. 18. & 49. 18.

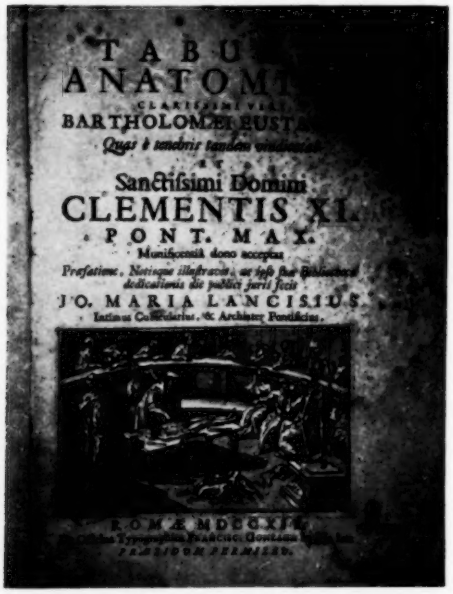
Musculus organo auditus adnexus, qui in maiori proprium officii malleo effectum inferius, 11. 15. & 51. 16 ; In cane est, non longe ab inferiore, glandulam cornuam dictam habet. 44. 16.

Figura

TABVLA SEPTIMA

FIG. 3 EXPLANATION OF THE ILLUSTRATIONS SHOWING THE OSSICLES IN TABULA SEPTIMA, OPUSCULA ANATOMICA.

"Two small shapes or figures relating to the organ of hearing; three small bones hidden in the cavity of the stone-like bone, the main muscle is adapted to their movement — in fact, to a higher degree in Man, while quite less so in a dog. A small, anvil-like bone — 8.16 and 48.19. A small bone which corresponds to the hammer — 9.15 and 49.20. A small bone, comparable with a so-called "stirrup" — 19.18 and 49.18 — overlooked (disregarded) by Vesalius. A muscle which every anatomician well knows, connecting with the major bone formation referred to as "the hammer." In the dog, however, a small horn-like gland is attached to it — 11.18 and 51.16."



TABU
ANATOM
CLARISSIMI VIRI
BARTHOLOMEI EUSTA
Quæ à tenebris tandem vindicata
ET
Sanctissimi Domini
CLEMENTIS XI.
PONT. MAX.
Munificentiâ dono accepta
Prefatione, Notisque illustrata, ac ipsius suæ Bibliothecæ dedicationis die publici iuris facta
JO. MARIA LANCISIUS
Intimus Cubicularius, & Archiater Pontificius.
ROMÆ MDCCXIV.
Typographia FRANCISCI Gonzagæ
PRÆLO HOC PERMISSU.

FIG. 4

TITLE PAGE OF THE TABULAE ANATOMICAE
"THE ANATOMICAL TABLES
of
EXCELLENCY
BARTHOLOMAEUS EUSTACHIUS

rescued from the darkness (of oblivion) and graciously accepted by virtue of the generous donation of His Holiness Clement XI, the Highest Pontiff, and published by Jo. Maria Lancisius, Personal Physician and Chamberlain of the Pope, with a Foreword, Notes and Dedications from his own Library

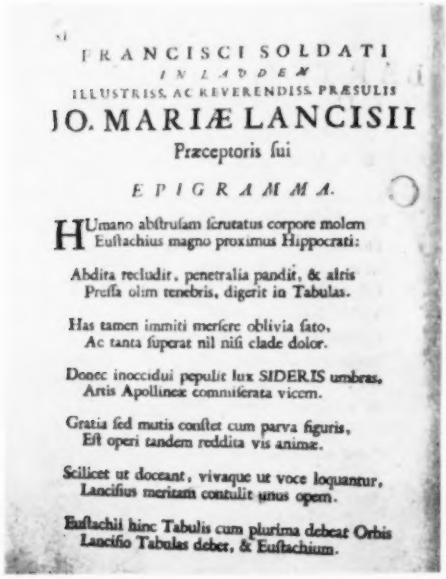
Rome, 1714.

Printing Shop of Franciscus Gonzage, on the Via Lata — with the permission of the Governors."

NOTE. — The signature of J. W. W. Werner may have been the Johann Wilhelm W. Werner born in 1715, and who became Professor of Medicine at the University at Halle. This may have been his book.

by the numbers on the border, both vertical and horizontal, intersecting them with a ruler, as one does on a map with the given coordinates.

In 1744, Bernardi Siegfried Albini[1] published a volume describing and explaining the same engravings, and it would seem from comparison of the plates that most of these illustrations were copies of the Tabulae Anatomicae of Lancisi. This volume of Albini was entitled, "Explicatio Tabularum Anatomicarum (of) Bartholomaei Eustachii" (Fig. 6). Albini used line drawings, with numbers, to indicate the various anatomical parts. The original Tabulae of Eustachius were copied and plagiarized, the last volume appearing in 1800. Copies of copies of the original Tabulae have been made, and many controversies regarding them have arisen. Had these plates appeared in 1552, when they were completed, Eustachius would have equal statue with Vesalius (circa 1514-1564) as one of the founders of Modern Anatomy.[17] The "Fabrica" of


FRANCISCI SOLDATI
IN LAUDEM
ILLUSTRISS. AC REVERENDISS. PRÆSULIS
JO. MARIÆ LANCISII
Præceptoris ſui

EPIGRAMMA.

HUmano abſtruſam ſcrutatus corpore molem
Euſtachius magno proximus Hippocrati:

Abdita recludit, penetralia pandit, & atris
Preſſa olim tenebris, digerit in Tabulas.

Has tamen inmiti merſere oblivia fato,
Ac tanta ſuperat nil niſi clade dolor.

Donec inoccidui pepulit lux SIDERIS umbras,
Artis Apollineæ commiſerata vicem.

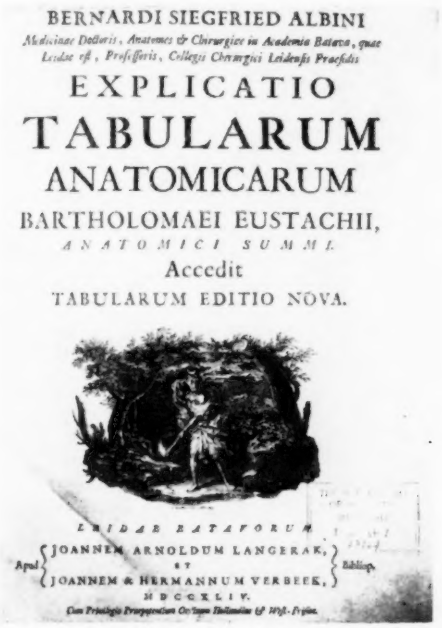
Gratia ſed mutis conſtet cum parva figuris,
Eſt operi tandem reddita vis animæ.

Scilicet ut doceant, vivaque ut voce loquantur,
Lancifius meritam contulit unus opem.

Euſtachii hinc Tabulis cum plurima debeat Orbis
Lancifio Tabulas debet, & Euſtachium.

FIG. 5
PAGE 40, LANCISI, TABULAE ANATOMICAE.
"EPIGRAM
of *Franciscus Soldatus*,
in honor of his teacher,
the Illustrious and Reverend President
JO. MARIA LANCISIUS.

Eustachius, who is next to the great Hippocrates, he who has investigated the abstruse mass in the human body, reveals hidden secrets, unfolds the innards and orders into Tables that which once was in darkness. Yet, harsh fate has made these Tables sink into oblivion, and so much loss does not even exceed the suffering. Until the light of the never-setting SUN dispersed the shadows, having taken pity on the vicissitudes of the Apollonean Art. Yet, small drawings do little good, and at last the vital strength has been restored to the work. Let them teach, let them speak aloud, a Lancisius has brought us a meritorious work. Hence, let the world owe a debt of great gratitude to the Eustachian Tables; to Lancisius it owes the Tables — and Eustachius."


BERNARDI SIEGFRIED ALBINI
Medicinæ Doctoris, Anatomes & Chirurgiæ in Academia Batava, quæ Leidæ eſt, Profeſſoris, Collegii Chirurgici Leidenſis Præſidis

EXPLICATIO
TABULARUM
ANATOMICARUM
BARTHOLOMAEI EUSTACHII,
ANATOMICI SUMMI.
Accedit
TABULARUM EDITIO NOVA.

LUGDUNI BATAVORUM
Apud { JOANNEM ARNOLDUM LANGERAK, ET JOANNEM & HERMANNUM VERBEEK, } Bibliop.
MDCCXLIV.
Cum Privilegio Præpotentium Ordinum Hollandiæ & Weſt-Frieſiæ.

FIG. 6
TITLE PAGE, EXPLICATIO
TABULARUM ANATOMICARUM.

"Explanation of the Anatomical Tables of the greatest anatomist, Bartholomaeus Eustachius by Bernardus Siegfried Albinus, Doctor of Medicine, Professor of Anatomy and Surgery at the Academy of Holland in Leyden, President of the Surgical College of Leyden. Ninth Edition of the Tables. Leyden, Holland. Printed by Joannes Arnoldus Langerak and Joannes Hermann Verbeck, Printers. 1744."

Vesalius is to this day a wonderful and superlative collection of illustrations of human anatomy but in comparison with Eustachius, Vesalius is not so accurate. In the multitude of discoveries, Eu-

stachuis has only Vesalius and Leonardo da Vinci as superiors. Unfortunately, the Tabulae did not appear until over 140 years later. Even then, they were hailed as masterpieces. To a great extent, they were excellent for teaching purposes. Aside from the general anatomy depicted in the 47 Tabulae, and only seven of which had accompanying descriptions by Eustachius, there were several very special studies made by him.

A meager description[9] of the tuba auditiva is found in the Opuscula in a chapter in that work. It is really a letter addressed to Francesco Alciato, dated October, 1562, from Rome. No illustration of the tube is found in any of the Tabulae Anatomicae. He also described the malleus,[10] incus and stapes, and in his Opuscula Anatomica compared that of the human ossicles with that of the dog. A description of the tensor tympani is also given, including its attachment to the major process of the malleus. These anatomical details are all carefully annotated with numbers of the upper, lower and side coordinates of the Tabula septima of the Opuscula Anatomica. There is no description of the Stapedius muscle.

Of course, it was known to the ancients, as stated by Aristotle,[20] that "there is no passage from the ear to the brain but there is one to the roof of the mouth." Alcmaem, who discovered the optic nerve and also the tuba auditiva, assumed from his dissections, which were done on goats, that they breathed through their ears. However, no one ever named the tube. Eustachius named the central bony column of the cochlea as the mediolus.[18]

Portal[13] in his Histoire de L'Anatomie states that "Eustache has made important discoveries about the ear. He is the first who knew the canal, which on one side opens into the tympanium and on the other side into the back part of the nostrils. He has discovered the malleus muscle (tensor tympani) and indicated the origin and course of the nerve which winds in the interior of the drum (chorda tympani). He also has given an exact description of the limacon (snail) cochlea. The canal of communication between the nose and the ear, he has said, has the form or shape of a quill. . . . Although Eustache could have claimed this discovery, as none of the ancient anatomists had spoken directly of this canal, he did not feel embarrassed to give it his name. Eustache has acquired an immortal reputation for himself and nobody has ever refuted that he did not discover the tube."

The controversy regarding the discovery of the stapes seems to have been decided by Portal in favor of Eustachius.

The illustration of the Tabulae septima of the Opuscula Anatomica is the only illustration that contains special anatomy that has been described by Eustachius (Fig. 3).

Portal states, "Eustache has mentioned three ossicles of hearing, 'le marteau, l'enclum and l'etrier.'[14] The first two were known by Achillinus and by de Berenger Carpi. As to the third (Eustache) attributes to himself. 'There are,' he says, 'today several contests on the discovery of this bone. Some contend that the Roman anatomists did not know about it and attribute the discovery to Ingrassius, the celebrated Sicilian physician and philosopher. But whomever one might give the credit of this discovery, I (Eustache) testify that before any one had spoken of it, wrote or published their works, I showed it to several persons in Rome and had it engraved on copper.' " (Fig. 3.) Portal continues: "Realdus Columbus (circa 1516-1559) also claimed the discovery of the stapes."

Realdus Columbus was the successor of Vesalius and wrote a textbook on anatomy that was published posthum-

ously, and was extensively used. He, it was, who described the turbinates and the mediastinum. Also, Gabriel Fallopius (circa 1523-1562), the discoverer of the aquaeductus Fallopi (facial nerve canal); F. hiatus (for the petrosal branch of the vidian nerve); F. muscle (pyramidalis); F. tube (oviduct); F. ligament (round ligament); F. valve (ileocecal valve) and also claimed to have discovered the stapes.

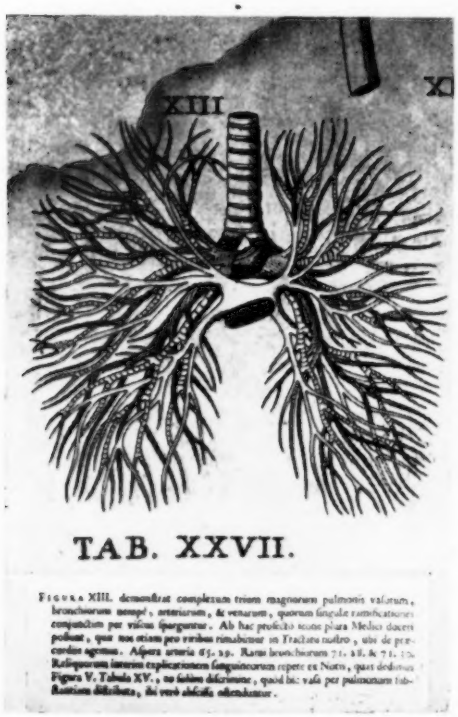


FIG. 7

TABULA 27, PAGE 66, LANCISI, TABULAE ANATOMICAE.

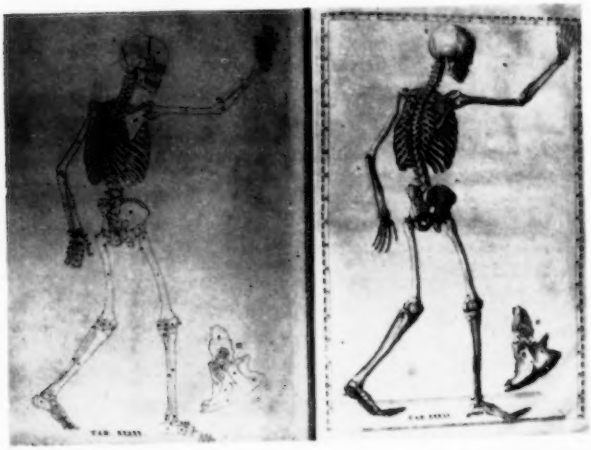
"Drawing XIII shows the relationship of three pulmonary vessels, as well as the bronchi, arteries and veins, the individual ramifications of which unitedly spread throughout the innards of the body. Medical men can learn much from this cross-section picture, which we shall also thoroughly investigate in the course of our treatise where we shall deal with the heart region. — The artery 65.29. The branches of the bronchi 71.28 and 71.30. In the meantime, review the explanation of the rest from the Notes given in Drawing V, Table XV, with the only distinction that while here the vessel is distributed throughout the lung substance, there separations are clearly evident."

Because of these varying and controversial statements, Portal appears to be in a quandary, asking the reader "which one shall we believe?" He concludes by saying "If we can judge by the prodigious research that Eustache has made on the organ of hearing, he must be looked upon as the discoverer."[15]

In the Tabulae Anatomicae of Lancisi, Tab. XXVII, XIII shows the trachea and bronchi with accompanying pulmonary veins and arteries.

The trachea is shown with nine rings, Fig. 7, and it would seem as though the general distribution of the bronchial tree was about equal in the right and left lung. The illustration also shows an equal sized right and left main bronchus. Unfortunately, it was unknown at that time that Leonardo da Vinci (1452-1519)[11] had shown split sections of the body in his anatomical drawings. If this advance in anatomy had been known by Eustachius and Vesalius, it would have been of great interest to have compared their illustrations with our modern ones. In Figs. 8, 9 and 10 will be found anatomical drawings by Eustachius that are of interest to otolaryngologists.

There exists in Sanseverino a marble bust of Eustachius (Fig. 11) made by Ercole Rosa. There is also another rather idealized one[2] in Rome. The former shows a strong featured man with a prominent nose, deep-set eyes and a long beard. He is clothed with an academic cap and gown that has a fur collar on it and puffed sleeves in the then prevailing style of professional men. He also is invested with a medallion hanging from a chain around his neck. One would conclude that most likely this effigy represents him toward the end of his life, and he would seem to be a man over 50 years of age. In the engraving that illustrates the frontisplate in Lancisi Tabulae Anatomicae of Eustachii, it shows a demonstration of anatomy by



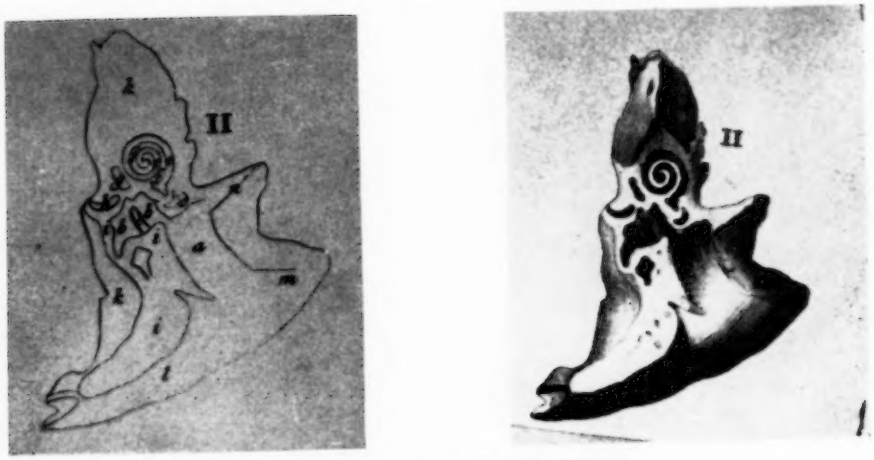


FIGURA SECUNDA.

Os temporis dextrum, d quo refectum os squamosum, refecta pars ... pori acustici, apertum tympanum, effracta per longitudinem ... effractus canalis semicircularis superior idemque minor.

a Porus acusticus.

bb Tympanum.

c Fenestra ovalis.

Foramen, ex triangulum effectum praeter Du Aud. org.

d Fenestra rotunda ut videtur.

e e e Cochlea, cc hinc parte effracta. Effracta pars ut principio cochleae ... amplior exhibita, & tanquam in duos angulos abeuns: quarum ... alterum, qui fenestrae rotundae d propior, cochleae cavum ad fenestram illam pertinet: juxta alterum autem, pertinet ad vestibulum. De cochlea vide Epist. de Aud. organ.

g An introitus aquaeductus Falloppii? qui introitus in infantibus dura mater superinducta claudatur; postea autem superincrescente squama ossea.

h Canalis semicircularis superior idemque minor.

iii Hinc resectum os squamosum.

k k Os petrosum. In quo à dextro latere interae k superioris, expressa pars canalis, per quem arteria carotis interna & nervi intercostalis tum in cranium penetrant.

l Os mammillare.

m Processus mamillaris.

n Squama, quae processui styliformi ossis temporis affidet.

FIG. 8

FIG. 8

TABULA 45, ALBINI, EXPLICATIO TABULARUM ANATOMICARUM (Second Figure).

"Right temporal bone.

The squamous part has been removed and also the anterior part of the external auditory canal, the tympanum is exposed, showing a longitudinal section of the cochlea, also the superior semicircular canal

a.	Ex. aud. canal	g.	Aquaeductus Fallopio
b.b.	Tympanum	h.	Superior semicircular canal
c.	Fenestra ovalis	III.	Section of squamous bone (process)
d.	Fenestra rotunda	k.k.	Petrous bone (process)
e.e.		l.	Mastoid
and	Cochlea	m.	Mastoid tip
f.f.		n.	Squamous bone (process)."

This illustration of the temporal bone in Tab. 45 of Albini is a facsimile of the Lancisi engravings and a typical one in explaining the anatomical detail. Albini published his interpretation of the copper engravings of Eustachius thirty years after Lancisi. They are apparently copies of the Lancisi engravings and not of the original copper plates of Eustachius. Albini used a line drawing with numerals on opposite page to describe the illustration. In this particular illustration (Tab. 45 of the second figure), it is placed in the vertical plane. In order to understand the anatomical details better, turn the drawing into the horizontal plane and at an angle of forty-five degrees. There is some slight distortion in the prospective. The artists of that day had but two planes in their conception of art. It was not until some years later that the conception of depth generally was understood and used in illustrations.

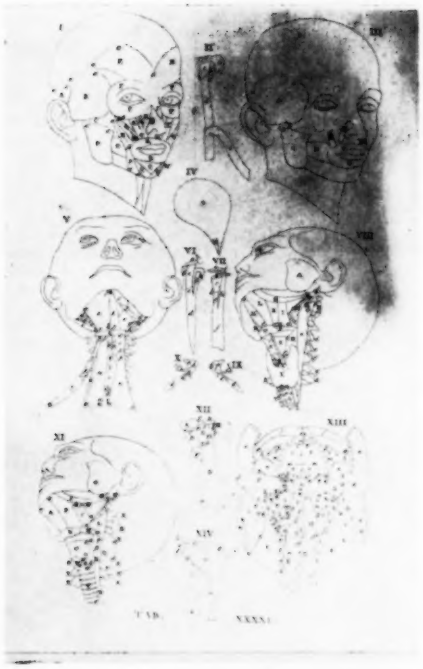
FIG. 9

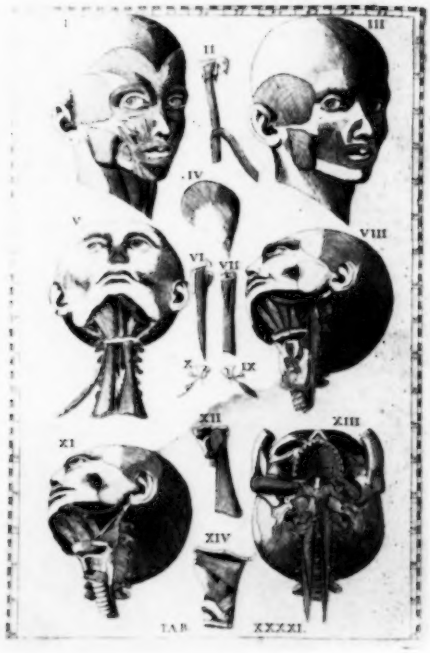
(A) EXPLANATION OF TABULA 42, LANCISI, TABULAE ANATOMICAE.

"Larynx with cartilages, muscles and one ventricular orifice, pharynx, hyoid bone and the tongue with various muscles."

(B) EXPLANATION OF TABULA 46, LANCISI, TABULAE ANATOMICAE.

The accessory pneumatic cavities (sinuses) are not shown. The lamina papyracea of the ethmoid is shown in Fig. I, also the nasal and lachrymal bones. The sella turcica is shown. The perpendicular plate of the ethmoid and the cribiform and the crista galli are meagerly shown. The maxillary sinus is shown in Tab. 47, the illustration showing it from the inner and posterior aspect. Tab. 46 is not illustrated in this article.





K. K Extrema partium offearum tubarum Euſtachianarum.

A caverna Oſſis lapidei, in quam Meatus Auditorius, Conchion appellatus, finitur, via in Narium cavitatem perforata eſt; ab illa cum Meatus alter oritur rotunda canaliculo ſimilis, & inſtar tenuioris calami amplus, qui oblique ad anterius interiusque baſis capitis latus procedens, in medio quatuor foraminum totum iſtud os penetrat atque perfodit K; nam poſterioris ipſius ſede arteria ſoporaria Calvariam ingreditur P; anterioris quartum nervorum Cerebri jugum extra ipſum emergit H; externum latus arteriae in duram Cerebri membranam diſtributae oſſium gratificat I; internum denique fiſſura quaedam circumſcribit, quae à cuneum referentis ac lapidei Oſſis extremis partibus, obliqueque infra & ante dudtii, ſit M. De Aud. org.

T I Ri.

Fig. 10

TABULA 41, FIG. 13. ALBINI, EXPLICATIO TABULARUM ANATOMICARUM.

"K. K. The Extremity of the Bony Parts of the Eustachian Tubes.

The way to the nasal cavity opens from the cavern of the stone-like bone in which the auditory passage called "Conchion" — ends. From this, in fact, starts another passage, like a small roundish channel, of which the width is similar to that of a thin reed; it proceeds at a slant, to the inner frontal side of the base of the skull, and this entire bone penetrates and comes through K in the middle of four holes; at the rear seat of it, the soporary artery enters the Calvaria P; at the front seat, the yoke of the four skull (brain) nerves emerges H beyond it; the external side of the artery spreads out in the hard membrane of the skull exposes the organ of hearing I; finally, the inner one is circumscribed by a certain fissure, which at the wedge and the extreme ends of the bone, at a slant below and before the ducts, becomes M. Organ of Hearing."

Note. — The "four holes" are interpreted to mean the carotid canal, the jugular fossae, the foramen spinosum and the foramen ovale; the "soporary" artery as the internal carotid and the "yoke of the four skull nerves" is interpreted to mean the branches of the trigeminal nerve and subdivisions of the mandibular or inferior maxillary branch. The "certain fissure" is the middle lacerated foramen.

FIG. 11
Bartolomeo Eustachi.

a Professor or Doctor in a "Theatrum Anatomicum." (Fig. 4.) The demonstrator is the only one present who has his hat on. The associates, assistants and others are uncovered. A lecture is being given on a prepared dissection and the demonstrator, whom we are to assume is Eustachius, is indicating by pointing with his index finger, probably to the heart. The instruments for dissection are on the table which is of special construction. These instruments shown would seem to be those used in conducting a necropsy, and it is conceded that Eustachius established this practice[5] in the Roman hospitals, not so much for anatomical purposes, but to determine the cause of death. This engraving was made by Pier Ghezzi (circa 1674-1755). He was an engraver whose finest work was done in architectural lines. He was not the engraver who made the Tabulae. They had been made by de Musi some 140 years previously. The date of his (Eustachius) birth is variously given as 1500 to 1520. He was a linguist and well versed in the Greek and Arabic languages. He became Professor of Medicine at the Studeo della Sapienza in Rome and understood the value and importance of anatomy and pathological anatomy. He[6] investigated the struc-

ture of the kidney, the teeth, the ossicles of the ear, the azygos vein, the ductus thoracicus, the valve in the vena cava inferior, the cranial nerves, the muscles of the head and neck and the valves of the coronary veins. His name has been perpetuated by the tuba auditiva (Eustachian tube) and the valve in the fetal heart, being a membranous fold in the right auricle between the mouth of the inferior vena cava and the auriculo ventricular aperture.

Most of Eustachius' work, anatomical, literary and philosophical, was done in Rome. It would seem as though he had had very little communication nor was he influenced by the Northern Italian schools, where Vesalius had produced his De Fabrica.[19]

Eustachius[7] was a great upholder of Galen, which would appear rather paradoxical in the light of his very accurate anatomical drawings. It would seem as though these illustrations would refute his Galenic tendencies. It must be remembered he was a physician in attendance at the Vatican (Pope Sixtus IV) and being the personal physician of Cardinal Rovere, later Pope Sixtus V. Galenic teachings and precepts were sacrosanct. It would require a strong-willed individual who would be willing to teach and expound their many fallacies, while still accepting the patronage of the Pontiff.

Whether or not Eustachius saw the De Fabrica of Vesalius, there is no record. However, it would seem that he knew about the teachings of Vesalius for he defended Galen or rather he justified the accepted attitude of the constituted clerical tradition and authority. As stated elsewhere, by his accurate drawings of the Tabulae he refuted these Teachings of Galen. But these Tabulae, with the exception of seven, were not published until 140 years after his death.

Vesalius refuted the contention that the structure of the human body had changed within the 1300 years, since the Teachings of Galen. In expiation for this heresy, he made a pilgrimage to Jerusalem to propitiate those in authority. On his return journey from the Holy Land, his ship was wrecked and his death was shrouded in mystery.[3]

To return to Eustachius, the implications are that he did not care nor dare to refute Galen and agree with Vesalius. They were both young men at the time. Vesalius, when he completed the De Fabrica, was 29 years old, and, of course, at that period Eustachius was only 23 years old. The distance from Rome to Padua took a week to travel and heretical teachings, while rapid in spreading, were nevertheless suppressed. Printed books were few and illustrated ones fewer.

In the introductory letter which Eustachius dedicates to his readers, he states that he has the greatest respect for the older authorities. (Fig. 12.) Nevertheless, he also remarks that he has discovered and compared them with the older and recent anatomists, and that he intends to show, in his "opus magnus," the dissensions and controversies among anatomists. He excuses himself because of his painful joints and advanced age — approximately 53 years. He died in his 54th year. The controversy was still raging 140 years later, as evidenced in the inclusion of the poem by Petrus Pinus of Urbini in the Lancisi Tabulae Anatomicae (Fig. 13).

Petrus Pinus was the "competent collaborator" he mentions in the letter. (Fig. 12.)

CONCLUSIONS

Eustachius was an anatomist of superior ability. Excepting for the seven illustrations in his Opuscula that were printed the year of his death, his great

BARTHOLOMÆVS
EVSTACHIVS

LECTORI.

TANTVM ſemper apud me valuit veterum ſcriptorum Authoritas; tantumque & ſtudij, & operæ, in veri inquiſitione inueſtigationeque ponendum eſſe exiſtimaui; vt ſummos in re medica viros, qui vere principes eius facultatis authores, & habentur, & ſunt; quos imitarer, & ſequerer Hippocratem, & Galenum, Duces in primis delegerim: ac in infinitorũ pene hominum, & eorum animantium, quæ in noſtra terra gignuntur, corporibus incidendis & diſſecandis, quod ſtudium ἀνατομὴν dicunt, multos iam annos diligentiſsime ſim verſatus. Quæ in hac veri inquirendi ratione a me reperta ſunt; ac ſummo ſtudio obſeruata; cum his, quæ veteres olim, & noſtri etiam Anatomici ſcripta reliquere, ſocio laborum adhibito Petro Matthæo Pino Vrbinati, accuratiſsime cõtuli. Itaq; factum eſt, vt ſex, & quadraginta tabulas æreas, in quibus noſtra hæc diligentia apparere facile poſsit; librumq; de diſſenſionibus, ac controuerſijs Anatomicis propediem ſim editurus. Quod certe multo prius feciſſem, niſi, & ingraueſcente iam ætate, & vehementiſsimo arthriculorum dolore, quo ſtudia retardantur, atq; fortunarum mearum imbecillitate, quæ ne tantum opus aggrederer ad imprimendum deterruit, fuiſſem prohibitus: voluntas certe non defuit, & animi ad id agendum propenſio. Tractationem autem hanc de Renibus, cæteraq; anatomica opuſcula, quæ nunc primùm edenda curaui, & libenter perlegas, & maturè iudices velim. Hæc enim cum probaueris, multo vtiliora poſt hac a me edenda, ut polliceri non dubito, ſic te lecturum tibi facile perſuaſeris. Vale.

* 2 in

FIG. 12

PAGES 2 AND 3 OF THE OPUSCULA ANATOMICA.

"BARTHOLOMAEUS EUSTACHIUS,

To the Reader.

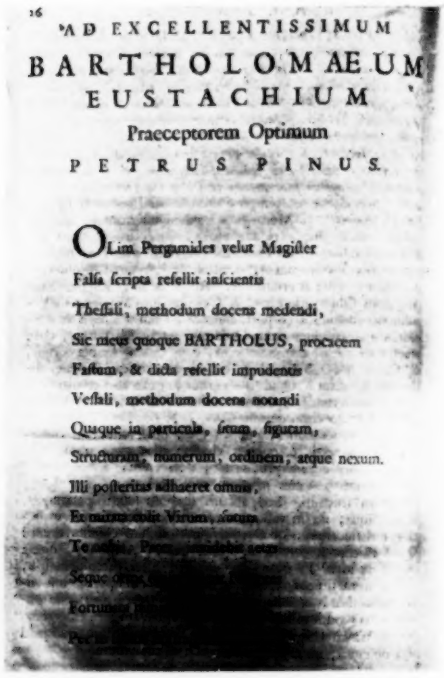
I have always held in great respect the authority of the old writings, studies and works; I have had the opinion that they indeed are to be studied and investigated, as well as the greatest men of the medical profession, its true creators, whom I should imitate and should follow Hippocrates and Galenus, whom I would call the greatest leaders; also for many years already I have been well versed in cutting and dissecting bodies of almost all kinds of humans and other living beings born onto this Earth, which science is called ANATOMY. In which study, many truths I have discovered and observed with utmost diligence; with these I have carefully compared that which is to be found in the writings of the ancient and also of our anatomicians, with the collaboration of the competent Petrus Matthaeus Pinus Urbinatus. Thus, it has been made possible that this endeavor of ours be shown in the data contained in forty-six Tables; soon I shall also publish a book on the dissensions and controversies among anatomicians. I certainly would have done this sooner, had it not been for my advanced age and violent gouty pains in my joints, which have delayed my investigations; also the stupidity of my fate has made it impossible for me to elaborate so much work and to have it ready for printing. Certainly, it was not any lack of intentions on my part, and I have every intention to do so. Now, reader, I want you freely to peruse this treatise about the kidneys, and also the other anatomical works which I have now had published. First of all, I would like to have mature judgment. You will indeed verify this, many useful things are still to be published by me, which I can safely promise, so that you may easily pursue this lecture. Farewell."

contribution to anatomy was not developed until nearly a century and a half afterwards. In this respect, he parallels Leonardo da Vinci, whose work was not rediscovered for over 400 years. Nevertheless, the Eustachian Tabulae were in considerable vogue in medical circles from 1714 to 1800. The accuracy of most of the illustrations in the Tabulae are such that they might be used even today. It is unfortunate that these Tabulae were lost at a time when they would have been so valuable to the medical student. The evidence of their value is manifest, for after 140 years, when they were recovered and released, they were used continuously for over 80 years in the teaching of anatomy.

Controversies of those days were bitter and should they invoke one with the Church, retribution quickly followed. These Tabulae may have been suppressed.

Eustachius was in the midst of the Vesalian discussion and while he did not repudiate Galen, his Tabulae Anatomicae definitely did so.

With but few exceptions, the brilliant men of that day rarely reached 50 years of life. Eustachius died when he was 54 years old, while on a journey from Rome. His name attached to the tuba auditiva is just one of the mysteries that occur in anatomical nomenclature and as advanced by Portal "because of his researches and knowledge of its anatomy, he was not embarrassed to attach his name to it."[14] This statement implies that Eustachius was cognizant of this. Regarding his knowledge of the function and physiology of the tube, very little can be said. A very careful translation of his Opuscula may reveal some further facts in this regard. In any event one can but admire this man for his diligence and the accomplishment in the making of the Tabulae Anatomicae.


AD EXCELLENTISSIMUM
BARTHOLOMAEUM
EUSTACHIUM
Praeceptorem Optimum
PETRUS PINUS.

OLim Pergamides velut Magiſter
Falſa ſcripta refellit inſcientis
Theſſali, methodum docens medendi,
Sic nicus quoque BARTHOLUS, procacem
Faſtum, & dicta refellit impudentis
Veſſali, methodum docens notandi
Quoque in particula, ſitum, figuram,
Structuram, numerum, ordinem, atque nexum.
Illi poſteritas adhaeret omnis,
Et mirans dicit Virum, Acutum
Te nobis, Pater, [illegible]
Seque dicit [illegible]
Fortunat[illegible]
Pe[illegible]

FIG. 13

PAGE 26, LANCISI, TABULAE ANATOMICAE.
"Petrus Pinus,
to that greatest of all teachers,
EXCELLENCY
BARTHOLOMAEUS EUSTACHIUS.

Once upon a time, Pergamides, as a Magister teaching how to heal, disproved the false writings of the ignorant Thessalius. The same way also my Bartholus, has rebutted the wanton pride and teachings of the impudent Vessalius, while teaching how to observe every single little piece, every location, shape, structure, number, order and connection. All posterity is his adherent and admiringly reveres this man. The future will envy us for you, Father, and the later generation will wish to have been born sooner. Our age will be praised as exceedingly happy and exceedingly lucky — on account of you."

Illustrations of accurate anatomical studies are of but recent date, certainly not over 400 years. If comparison is made of the Eustachian, Vesalian, Fallopian and other contemporary anatomical works, with those of the previous decades, one may easily understand the status of surgery and medicine of that period. (Fig. 14.)

Fallopius, Vesalius and Eustachius should be considered an immortal triad, whose life work was of such force that they reformed the study of anatomy into factual and accurate channels by repeated dissections. The acme of their ability was that they were able to illustrate and preserve their dissections by drawings so that the previously accepted inaccuracies were swept away and the true anatomy revealed.

Thanks and acknowledgement for the many courtesies extended to me are hereby made to Doctor Archibald Malloch, Librarian of the New York Academy of Medicine, and also to Miss G. L. Annan of the Rare Book Department of the Library.

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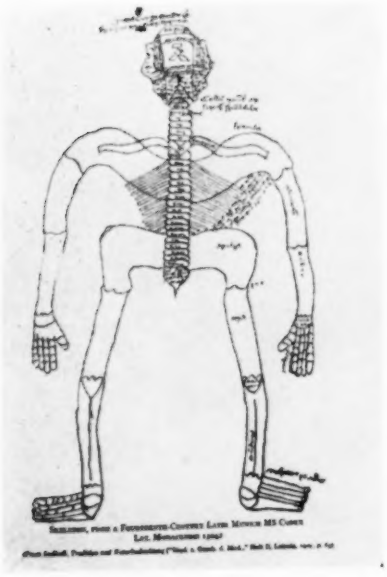

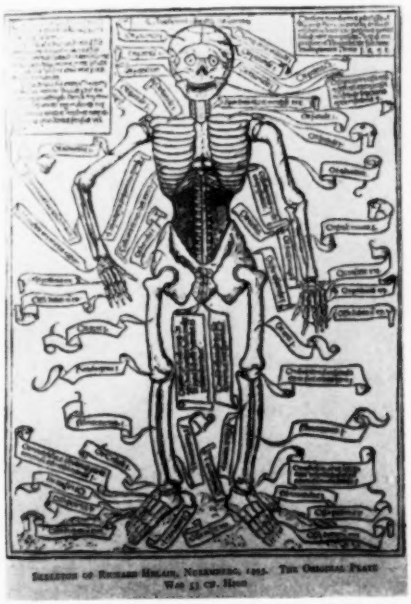

FIG. 14

These illustrations are taken from Choulant, pg. 69 and 71. The crudeness of these drawings is self-evident, and it would be difficult for the modern mind to conceive what amount of anatomy — general, topographical or surgical — could be gained from them.

Banner Presented to the Great Lakes Choir by the Academy, October 12, 1943
(Official U. S. Navy Photograph)

TRANSACTIONS
American Academy of Ophthalmology and Otolaryngology
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W. HOWARD MORRISON, Omaha, Nebraska, *Associate Editor*
HENRY L. WILLIAMS, Rochester, Minnesota, *Associate Editor*
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All communications regarding the TRANSACTIONS should be addressed to William L. Benedict, M.D., Executive Secretary-Treasurer, the American Academy of Ophthalmology and Otolaryngology, 100 First Avenue Building, Rochester, Minnesota

Editorial

The Bluejacket Choir

Only a few days before the opening of the convention, the Committee on Arrangements was ironing out the final wrinkles in the program for Monday and Tuesday evenings. Every detail must be set down and put in the hands of the hotel management, the director of the program and the officers of the convention. Final arrangements for the United States Naval Training Station Great Lakes Choir included transportation and meals for 45 sailors, time of appearance, length of program and time of dismissal. Sitting about the table were Chaplain Hanson, Dr. Walter Theobald, Richard Collison of the Palmer House, Lt. P. H. Faust, Dr. Harry S. Gradle and Dr. W. L. Benedict.

Chaplain Hanson told of the great number of engagements that were being filled by one or more of the six Great Lakes choirs organized and functioning through the Department of the Navy. His one regret was that the Bluejacket Choir had no banner of its own. He was told that if he could get official authorization for a banner, the Academy would furnish it with its compliments. Chaplain Hanson brought to the Academy banquet 45 of the top singers from the Naval Choir, selected from more than 6,000 auditions.

Authorization for the banner was soon obtained. Here is Chaplain Hanson's letter of appreciation:

U. S. Naval Training Station
Great Lakes, Illinois
29 October, 1943

W. L. Benedict, M.D.
100 First Avenue Building
Rochester, Minnesota

Dear Doctor Benedict:

Believe me, sir, we have never had more wonderful responses, both in letters and in personal conversation, than we have had concerning your banquet. You folks make us really feel that we are somebody. I want to thank you so very, very much for all you have done for the choir.

Now, in regard to the banner, I have called Doctor Theobald, and he advised me that it would be satisfactory if I would order it so that we might have it for Navy Day. I did just that, and we marched through all the loop with that flag out front all yesterday morning. It is a very beautiful banner, and we are very proud and happy to have it.

May I, in this manner, extend my personal and sincere thanks to you and the American Academy of Ophthalmology and Otolaryngology.

Very sincerely,

(Signed) HJALMAR F. HANSON
Chaplain, U. S. N. R.

Extracts from Letters to the Officers of the Academy

Dr. James A. Babbitt:

Judging from the attendance, the enthusiasm and the character of the papers, you had the most outstanding meeting the Academy ever has held.

Dr. James A. Babbitt:

I did enjoy the meeting of your organization. I have never before attended meetings that were intellectually so high brow and socially so fraternal as these were.

Dr. W. L. Benedict:

We were gratified at the ever-increasing interest of the members of the American Academy of Ophthalmology and Otolaryngology, which was evidenced by the very important volume of orders placed with our Company.

Dr. W. L. Benedict:

The members of the American Association of Orthoptic Technicians wish to express their appreciation for the privilege of attending the meeting of the American Academy of Ophthalmology and Otolaryngology, as the Academy's guests. The benefits derived from the lectures were both interesting and stimulating.

Professor Olof Larsell — Honorary Fellow

The Council of the American Academy of Ophthalmology and Otolaryngology recommended that Dean Olof Larsell, M.A., Ph.D., Sc.D., be made an Honorary Fellow of the Academy because of his distinguished services to otolaryngology, particularly in the fields of anatomy and neurology. He collaborated with Dr. Ralph A. Fenton at the University of Oregon Medical School on a series of experimental studies of tissue changes in the sinuses by vital staining under various degrees of irritation and of infection, and after exposure of such infected membranes to the usual therapeutic agents as well as to various sorts of chemical, hormonal and electro-physical stimuli, including the x-ray. After the initial study in 1931, the Research Committee of the Council of the Academy sponsored these studies, which were presented at Academy sessions until 1938. Later research included demonstrations of the pathway of referred pain from the sphenopalatine region through ischemia of the retro-auricular and lateral cervical area; and of the direct route of infection from the sinuses through the deep cervical and paralaryngeal glands into lymphatic trunks which drain directly into the pulmonary circulation and thus to the periphery of the lung.

Doctor Larsell's final Academy-sponsored research, on the development of cochlear areas as seen histologically after testing responses to sound stimuli in the pouched young of the opossum, was presented at the 1943 session.

Born in Sweden in 1886, brought to the West when a child, educated in Oregon and trained in 1913-14 as a neuro-anatomist by Dr. C. J. Herrick, he received his Ph.D. degree from Northwestern University Medical School in 1918, and has been Professor of Anatomy at the University of Oregon since 1921. In 1938 he became Dean of the Graduate Division of the entire system of higher education in Oregon. He is the author of a fine text book on neuro-anatomy and of a monumental descriptive monograph on the cerebellum, and the section on the nervous system in Morris' Human Anatomy. He is also an authority on medical history.

The Academy is glad to honor one of its most distinguished collaborators, the great neuro-anatomist, Olof Larsell.

The Constitution and By-Laws*

The Constitution and By-Laws of the Academy remained unaltered as published in the TRANSACTIONS for 1933 (p. 491-497) until revised by a special committee authorized by the Council October, 1942, and appointed by President Ralph I. Lloyd. The committee was composed of Dr. Carl H. McCaskey, Chairman, Dr. Frank E. Brawley and Dr. Sanford R. Gifford.

The committee held several meetings with the Executive Secretary-Treasurer for a critical review of the Council minutes for the years preceding and succeeding the year 1933. It had long been evident that the expansion of the Academy in membership and in activities since the publication of the Constitution and By-Laws 11 years ago had imposed new responsibilities in the officers and required provisions that were not then foreseen. While the Council has governed the Academy, since its reorganization in 1906, and through delegated authority has kept a guiding hand on all Academy activities, constitutional authority for Council action has never been specifically provided.

The application fee, fixed by the Constitution (Article III), has been doubled by Council action without change in the Constitution. In the same Article on Fellowship the Constitution reads, "An applicant for membership must be approved by the American Board for Ophthalmic Examinations or by the American Board of Examiners in Otolaryngology." Both of these Boards are now operating under different names.

In Article IV, under the heading "Officers," there is a provision for formation of a Council but the duties, powers and activities of the Council are not mentioned in either the Constitution or the By-Laws.

The annual report of the Executive Secretary-Treasurer shows considerable property in the form of stocks, bonds and fixtures. The By-Laws provide that the Comptroller "shall be the financial officer of the Academy, representing and responsible to the Council . . . shall supervise the work done in the Executive Secretary-Treasurer's office." This arrangement was satisfactory for only a few years. As a matter of fact the provision has never been strictly adhered to and is not now feasible. After a study of the aims and purposes of the Academy, with advice and counsel from bank and trust officers and attorneys, a new system of financial management was set up. Consequently the Committee on the Revision of the Constitution and By-Laws has proposed that certain revisions be made in the Constitution and By-Laws to give a sound structure to the Academy based on constitutional authority.

The proposed revisions of the Constitution are presented officially in the minutes of the annual business meeting for 1943, published in this issue of the TRANSACTIONS, and will be voted on as amendments in 1944.

Revision of the By-Laws as proposed by the committee was accepted by the Council and approved by unanimous vote of the Fellows at the business meeting October 12, 1943. The new By-Laws are now in force. While during the year 1943-1944, between annual business meetings, there appear discrepancies, these will be ironed out when the proposed amendments to the Constitution are adopted next year, as they undoubtedly will be. In the meantime the officers of the Academy are carrying on under the direction of the Council as in the past few years.

The committee wishes to acknowledge with thanks the services of Dansingburg and Plemling, attorneys, and Mr. Edw. A. Wobschall of the firm of Shannon and Byers, certified public accountants.

* Contained on pages 90 and 111.

Council Minutes

Forty-Eighth Annual Session

October 10-13, 1943

Palmer House — Chicago

SUNDAY, OCTOBER 10, 1943

Present:

James A. Babbitt, Lawrence T. Post, Sanford R. Gifford, William A. Krieger, Frank E. Brawley, Frank R. Spencer, Ralph I. Lloyd, Albert C. Snell, Carl H. McCaskey, William L. Benedict, Algernon B. Reese, O. E. Van Alyea, A. D. Ruedemann, Dean M. Lierle, Erling W. Hansen, Edward C. Ellett.

The meeting was called to order by President Babbitt at 10 a.m.

I

The Executive Secretary presented communications for Council consideration.

1. Report entitled "A Basic Industrial Visual Program" prepared by the Joint Committee of Industrial Ophthalmology.

Motion made by Dr. Lawrence T. Post, seconded by Dr. Frank R. Spencer that the report submitted be published as a supplement to the TRANSACTIONS. Carried.

2. "Manual for Program Outline for Rehabilitation of Aural Casualties of the War," prepared by Drs. Walter Hughson and Harold Westlake.

Dr. Dean M. Lierle recommended that the manual be published as a supplement to the TRANSACTIONS.

Moved by Dr. Carl H. McCaskey, seconded by Dr. Frank R. Spencer that the Manual be published as a supplement to the TRANSACTIONS. Carried.

3.

4. Film "Sound and Hearing," prepared by Dr. Eugene Lewis.

Moved by Dr. William L. Benedict, seconded by Dr. Frank R. Spencer that the Academy not purchase the film. Carried.

5. "Eye Exercises vs. Refractive Errors." Special reference to advertisements appearing in popular magazines concerning the correction of ocular disease by exercises instead of glasses.

Motion made by Dr. Lawrence T. Post, seconded by Dr. Albert C. Snell that this matter be referred to the American Committee on Optics and Visual Physiology. Carried.

6. Appointment of nominee from the Academy to the Board of Governors of the American College of Surgeons.

Moved by Dr. Ralph I. Lloyd, seconded by Dr. Lawrence T. Post that the Executive Secretary of the Academy continue to be the representative to this Board. Carried.

7. Resignation of Dr. Secord H. Large as Comptroller of the Academy.

Moved by Dr. O. E. Van Alyea, seconded by Dr. Sanford R. Gifford that no action be taken on Doctor Large's resignation. Carried.

8.

9. Publication of a volume containing a review of literature in otolaryngology during the war period. No action taken.

10. Incorporation of Academy.

Moved by Dr. William L. Benedict, seconded by Dr. Ralph I. Lloyd that the Academy continue as an inincorporated organization. Carried.

11. A Committee appointed by the Council in 1941 to investigate material of Lindsay Johnson.

Moved by Dr. William L. Benedict, seconded by Dr. Frank E. Brawley that the Committee be discontinued. Carried.

12. Regrets were read from Dr. Wesley Bowers, New York, N. Y., second vice president, concerning his inability to attend Council sessions because of illness.

II

Report of Executive Secretary on Fellowship

1. SEPTEMBER 1, 1943		
Honorary Fellows		20
Active Fellows		
Life		248
Senior (dues paid)	181	
Senior (delinquent)	3	
	184	184

Some items are omitted from this report. Complete Minutes are on file in the office of the Executive Secretary-Treasurer.

Junior (dues paid)	2,683	
Junior (delinquent)	189	
	2,872	2,872
Fellows excused from payment of dues		11
TOTAL FELLOWSHIP Sept. 1, 1943		3,335

2. The Executive Secretary recommended that the following Fellows be excused from paying dues: Dr. A. H. Cecha, prisoner of war; Dr. C. H. Fox, Denver, Colorado; Dr. L. L. Herriman, Alamose, Colorado; Dr. B. F. Hodsdon, Miami, Florida.

Approved by Council action.

3. The Executive Secretary presented a list of 22 names of Fellows who are in arrears for more than three years.

By Council action 19 were dropped from Fellowship in the Academy because of non-payment of dues.

4. Resignations have been received from the following Fellows:

Dr. Charles T. Estabrook, Worcester, Mass.
Dr. Rene H. Huvelle, Dallas, Texas
Dr. R. E. Robinson, Waverly, Iowa
Dr. Van Rensselaer Voislawsky, New York, N. Y.

The resignations were accepted by Council action.

5. Fellows deceased — 36.
John Finch Barnhill, Miami Beach, Fla.
Joseph C. Beck, Chicago, Ill.
Theodore S. Blakesley, Kansas City, Mo.
Donald Malcolm Campbell, Detroit, Mich.
Ewing Wilbur Day, Pittsburgh, Pa.
Joseph F. Duane, Peoria, Illinois
John Daniel Fouts, Dayton, Ohio
Arthur Henry Geiger, Chicago, Ill.
Allen Greenwood, Boston, Mass.
John Milton Griscom, Moorestown, N. J.
Will C. Harris, Cincinnati, Ohio
Sergius M. Ingerman, New York, N. Y.
Edward Jackson, Denver, Colorado
Wilson Johnston, Portland, Oregon
Robert McKee Lapsley, Keokuk, Iowa
Jonathon Brown Loring, Chicago, Ill.
S. Hanford McKee, Montreal, Canada
Willard Geist Mengel, Camden, N. J.
Stewart Brown Muncaster, Washington, D. C.
Eugene P. Norwood, Corsicana, Texas
Samuel Poindexter Oast, New York, N. Y.
Luther Crouse Peter, Philadelphia, Pa.
Grover Hewitt Poos, Palm Springs, Calif.
Howard Thomas Robinson, Cumberland, Md.
Joseph M. Rosenthal, Philadelphia, Pa.
Abraham Noah Schiller, New York, N. Y.
Louis Joseph Sebert, Toronto, Canada
Walter J. Sener, Media, Pa.
Frederick Casimir Simon, St. Louis, Mo.
Harry Bayliss Stokes, Omaha, Nebraska
James Craig Strong, Leadville, Colorado
Edward Franklin Stroud, Corpus Christi, Texas
Frank Vinsonhaler, Little Rock, Arkansas
Carl B. Wagner, Chicago, Ill.
C. B. Walker, Los Angeles, California
Walter Joseph Wherry, Omaha, Nebraska

6. SUMMARY OF FELLOWS:

Active Fellows, Sept. 1, 1943	3,335	
Excused from paying dues, Council action, 1943	4	
Deferred action (delinquent) Council action, 1943	3	
	3,342	3,342
Elected to Fellowship at annual business meeting, 1943	219	219
TOTAL FELLOWSHIP Oct. 12, 1943		3,561
Resignations 1943	4	
Deaths 1943	36	
Dropped for non-payment of dues 1943 Council action	19	
(Deducted from above count of Active Fellows)	59	

7. SUMMARY OF CANDIDATES:

Complete candidates	2
Incomplete candidates	13

III

Report of the Treasurer

The Treasurer presented the report on Examination of Accounts for the year ended August 31, 1943.

(See report in the minutes of the annual business meeting.)

IV

MONDAY, OCTOBER 11, 1943

Present:

James A. Babbitt, Lawrence T. Post, Sanford R. Gifford, William A. Krieger, Frank E. Brawley, Frank R. Spencer, Ralph I. Lloyd, Albert C. Snell, J. Mackenzie Brown, Derrick Vail, C. H. McCaskey, William L. Benedict, Algernon B. Reese, O. E. Van Alyea, A. D. Ruedemann, Dean M. Lierle, Erling W. Hansen, Edward C. Ellett.

The meeting was called to order by President Babbitt at 7:30 a.m.

V

Report of Committee on the Revision of the Constitution and By-Laws.

Moved by Dr. Ralph I. Lloyd, seconded by Dr. C. H. McCaskey that the revision of the Constitution as read by the Council be approved and offered at the annual business meeting. Carried.

Moved by Dr. C. H. McCaskey, seconded by Dr. Ralph I. Lloyd that the revision of the By-Laws as read by the Council be approved and offered at the annual business meeting for action. Carried.

VI

Report of Activities Committee

(Full reports of the Standing and Joint Committees are printed in full in the report of the business meeting.)

Standing Committees

Report of the American Board of Ophthalmology. Moved by Dr. Albert C. Snell, seconded by Dr. C. H. McCaskey that the report be received. Carried.

Report of the American Board of Otolaryngology. Moved by Dr. Sanford R. Gifford, seconded by Dr. Lawrence T. Post that the report be received. Carried.

Report of the Registry of Ophthalmic Pathology. Moved by Dr. Frank R. Spencer, seconded by Dr. C. H. McCaskey that the report be received. Carried.

Report of the Registry of Otolaryngic Pathology. Moved by Dr. Ralph I. Lloyd, seconded by Dr. Frank E. Brawley that the report be received. Carried.

Report of the War Activities Committee. Moved by Dr. Frank R. Spencer, seconded by Dr. Sanford R. Gifford that the report be received and the Committee discontinued. Carried.

Report of the Committee on the Conservation of Hearing. Moved by Dr. Frank R. Spencer, seconded by Dr. C. H. McCaskey that the report be approved. Carried.

Report of the American Association for the Advancement of Science. Moved by Dr. Sanford R. Gifford, seconded by Dr. Lawrence T. Post that the report be received and the Committee continued. Carried.

Scientific Exhibit. No report received. Moved by Dr. Ralph I. Lloyd, seconded by Dr. Albert C. Snell that the Committee be continued. Carried.

Report of Home Study Courses. Moved by Dr. A. C. Snell, seconded by Dr. J. Mackenzie Brown that the report be received. Carried.

Moved by Dr. Ralph I. Lloyd, seconded by Dr. William A. Krieger that the fee remain $10.00 for the duration.

Report of the Committee on the Standardization of Tonometers. Moved by Dr. A. C. Snell, seconded by Dr. William A. Krieger that the report be accepted. Carried.

Dr. Ralph I. Lloyd suggested that in accepting this report Dr. Jonas Friedenwald be told that the Council considers the work highly favorable and should be carried on to a practical conclusion.

Report of the Committee on the W. P. Wherry Memorial. Moved by Dr. Sanford R. Gifford, seconded by Dr. Ralph I. Lloyd that the report be accepted and recommendations approved. Carried.

Report of the Committee on the Pan-American Congress of Otolaryngology. Moved by Dr. Albert C. Snell, seconded by Dr. C. H. McCaskey that the report be received and placed on file. Carried.

Moved by Dr. William L. Benedict, seconded by Dr. Ralph I. Lloyd that the report be published as a supplement to the TRANSACTIONS. Carried.

Joint Committees

Report of the American Committee on Optics and Visual Physiology. Moved by Dr. Albert C. Snell, seconded by Dr. Sanford R. Gifford that the report be received. Carried.

A report was not received from the Eye Health Committee of the Student Health Association. Moved by Dr. Albert C. Snell, seconded by Dr. William L. Benedict that the Committee be retained. Carried.

Report of the Committee on Orthoptics. Recommendations: Continuation of active sponsorship by the Academy of the Orthoptic Council; repetition of annual grant of $100.00; re-appointment of Dr. John B. Hitz to serve for three years as the Academy representative. Moved by Dr. A. C. Snell, seconded by Dr. Sanford R. Gifford that the report be received and the recommendations approved. Carried.

Report of the Joint Committee of Industrial Ophthalmology. Moved by Dr. Albert C. Snell, seconded by Dr. C. H. McCaskey that the report be received and the Committee retained. Carried.

TUESDAY, OCTOBER 12, 1943

Present:

James A. Babbitt, Lawrence T. Post, Sanford R. Gifford, William A. Krieger, Frank E. Brawley, Frank R. Spencer, Ralph I. Lloyd, Albert C. Snell, J. Mackenzie Brown, Derrick Vail, C. H. McCaskey, William L. Benedict, Algernon B. Reese, O. E. Van Alyea, A. D. Ruedemann, Dean M. Lierle, Erling W. Hansen, Edward C. Ellett.

The meeting was called to order by President Babbitt at 7:30 a.m.

VII

Report of the Executive, Research and Finance Committee

1. Candidates for Fellowship (219). The names of 216 candidates were published in the July-August, 1943, issue of the TRANSACTIONS. Five candidates have since passed the Board examinations and two candidates died. (List published in report of annual business meeting.)

List of candidates approved by Council.

2.

3. Annual dues and entrance fee — 1944.

The Committee recommends that the entrance fee remain at $20.00, dues for Junior Fellows $10.00, dues for Senior Fellows $5.00 and dues for Fellows in military service $5.00. The Committee recommends that Life Fellows and Honorary Fellows continue to be exempt from paying dues.

Moved by Dr. Ralph I. Lloyd, seconded by Dr. Frank E. Brawley that these recommendations be approved. Carried.

4. The Committee recommends the re-appointment of Dr. William L. Benedict Editor-in-Chief of the TRANSACTIONS. It recommends that the Editor-in-Chief be permitted to appoint an associate editor in otolaryngology and an associate editor in ophthalmology.

Moved by Dr. Albert C. Snell, seconded by Dr. C. H. McCaskey that the recommendations be accepted. Carried.

5. The Committee reports the following appointments by the Editor-in-Chief of the TRANSACTIONS: Dr. W. Howard Morrison, Associate Editor for Ophthalmology and Dr. Henry L. Williams, Associate Editor for Otolaryngology.

Moved by Dr. James A. Babbitt, seconded by Dr. Frank R. Spencer that the appointments be approved. Carried.

6. Nomination of officers.

President.....Dr. Lawrence T. Post
St. Louis, Missouri

President-Elect .....Dr. Gordon B. New
Rochester, Minnesota

First Vice President....Dr. C. Stewart Nash
Rochester, N. Y.

Second Vice Pres....Dr. Harold F. Whalman
Los Angeles, California

Third Vice Pres.....Dr. Chevalier L. Jackson
Philadelphia, Pa.

Executive Sec'y-Treas....Dr. W. L. Benedict
Rochester, Minnesota

Secretary for Oto.....Dr. O. E. Van Alyea
Chicago, Ill.

Secretary for Oph.....Dr. Algernon B. Reese
New York, N. Y.

Sec'y for Instruction—
Ophthalmology....Dr. A. D. Ruedemann
Cleveland, Ohio

Sec'y for Instruction — Oto. and
Maxillofacial Surgery..Dr. Dean M. Lierle
Iowa City, Iowa

Secretary for
Public Relations....Dr. Erling W. Hansen
Minneapolis, Minnesota

Secretary for
Home Study Courses..Dr. Harry S. Gradle
Chicago, Ill.

Member of Council
(1947) .....Dr. James M. Robb
Detroit, Mich.

Nominations to Standing and Joint Committees:

American Board
of Ophthalmology.Dr. Frederick C. Cordes
San Francisco, Calif. — (4 years)
Dr. Everett L. Goar
Houston, Texas (1 year)

American Board
of Otolaryngology .....Unchanged
Ophthalmic Pathology .....Unchanged
Otolaryngic Pathology.....Unchanged
Conservation of Hearing.....Unchanged
American Association for the
Advancement of Science .....Unchanged
Scientific Exhibit .....Unchanged
Standardization of
Tonometers.....Drs. Cordes and McLean
(New members added to Committee)
W. P. Wherry Memorial.....Unchanged
Revision of
Constitution and By-Laws.....Unchanged
Pan-American Congress
of Otolaryngology .....Unchanged
(Dr. William L. Benedict and Dr. Lawrence T. Post to be ex-officio members.)

American Committee on Optics
and Visual Physiology.....No change
Eye Health Committee of
American Student Health Ass'n. No change
American Orthoptic Council.....No change
Industrial Ophthalmology .....No change

Moved by Dr. Ralph I. Lloyd, seconded by Dr. James A. Babbitt that the nominations be accepted. Carried.

7. The Committee recommends the nomination of Dr. Ralph A. Fenton, Portland, Oregon, to Life Fellowship in the Academy.

The nomination was accepted by the Council.

8. The Committee recommends the nomination of Professor Olof Larsell, Ph.D., Sc.D., of the University of Oregon, Portland, Oregon, to Honorary Fellowship in the Academy.

The nomination was accepted by the Council.

9. The Committee recommends that the 1944 convention be held in Chicago October 8-12, the final decision to be left to the discretion of the Program Committee at its January meeting.

Approved by the Council.

10. The Committee authorizes the Executive Secretary to take public liability and compensation insurance and Fidelity insurance bonds for Academy employees.

Approved by the Council

VIII

Report of Committee on Research and Finance (Sub-Committee of Executive, Research and Finance Committee)

1. The Committee recommends an appropriation of $1,500.00 for 1944 to the Registry of Pathology.

Moved by Dr. Ralph I. Lloyd, seconded by Dr. C. H. McCaskey that the appropriation be approved. Carried.

2. The Committee recommends the appropriation of $100.00 for 1944 to the American Orthoptic Council.

Moved by Dr. O. E. Van Alyea, seconded by Dr. Sanford R. Gifford that the appropriation be approved. Carried.

3. The Committee recommends the appropriation of $1,500.00 for 1944 to the Committee on the Conservation of Hearing.

Moved by Dr. Frank R. Spencer, seconded by Dr. C. H. McCaskey that the appropriation be approved. Carried.

4. The Committee recommends the appropriation of $50.00 for 1944 to the American Committee on Optics and Visual Physiology.

Moved by Dr. Albert C. Snell, seconded by Dr. Frank R. Spencer that the appropriation be approved. Carried.

5. The Committee recommends an appropriation of $2,100.00 for general secretarial expense.

Moved by Dr. Ralph I. Lloyd, seconded by Dr. Lawrence T. Post that this recommendation be approved. Carried.

6.

7. The Committee presented a resolution authorizing the Executive Secretary-Treasurer to enter into a custodian agreement with the First National Bank of Minneapolis for the purpose of custody of certain property.

Moved by Dr. Ralph I. Lloyd, seconded by Dr. Sanford R. Gifford that the authorization be given. Carried.

IX

Moved by Dr. Ralph I. Lloyd, seconded by Dr. C. H. McCaskey that Atlases, monographs and supplements to the TRANSACTIONS distributed by the Academy be sent to Colonel Derrick Vail gratis for distribution among military hospital libraries in England, Colonel Vail to designate which libraries should be the recipients. Carried.

WEDNESDAY, OCTOBER 13, 1943

Present:

James A. Babbitt, Lawrence T. Post, Sanford R. Gifford, William A. Krieger, Frank E. Brawley, Frank R. Spencer, Ralph I. Lloyd, Albert C. Snell, Derrick Vail, C. H. McCaskey, William L. Benedict, A. D. Ruedemann, Dean M. Lierle, Erling W. Hansen, O. E. Van Alyea, Algernon B. Reese, Edward C. Ellett.

Guests:

Gordon B. New, James M. Robb, Jonas Friedenwald, Harry S. Gradle.

The meeting was called to order by President Babbitt at 7:30 a.m.

X

Dr. Jonas Friedenwald, Chairman of the Committee on the Standardization of Tonometers, had been invited to attend the Council meeting to make further comment on the work of his Committee.

On behalf of the Committee on Standardization of Tonometers, Doctor Friedenwald asked for a sum not to exceed $500.00 for the establishment of a tonometric station in California.

Moved by Dr. Albert C. Snell, seconded by Dr. Sanford R. Gifford that the Council approve the appropriation of not to exceed $500.00 for the establishment of a tonometric station in California. Carried.

Moved by Dr. Albert C. Snell, seconded by Dr. Ralph I. Lloyd that the Council authorize the Committee on Standardization of Tonometers to certify in the name of the Academy instruments that conform to the standards laid down by the Committee. Carried.

XI

Honor Section

Dr. E. C. Ellett reported that a committee was appointed to make the awards for scientific exhibits. Awards made were as follows: Originality in Ophthalmology — Dr. A. D. Ruedemann; Originality in Otolaryngology — Dr. J. R. Lindsay, Exhibitor, and Dr. H. B. Perlman, Co-Exhibitor; Greatest Value to Medicine — Ophthalmology — Dr. Roy O. Riser; Greatest Value to Medicine — Otolaryngology — Committee on the Conservation of Hearing; Excellence in Presentation — Ophthalmology — Col. J. E. Ash, Exhibitor, and Helenor Campbell Wilder, Co-Exhibitor; Excellence in Presentation — Otolaryngology — Dr. Samuel Fomon, Exhibitor, Dr. Romeo A. Luongo, Co-Exhibitor; Dr. Morris S. Bender, Co-Exhibitor and Dr. Alfred Schattner, Co-Exhibitor.

Honor keys for 1943 have been awarded to Dr. A. W. Proetz, Dr. Gabriel Tucker, Dr. Perry Goldsmith, Dr. George M. Coates and Colonel J. E. Ash.

Dr. Thomas E. Carmody was elected President of the Honor Section for 1944, Dr. Frank E. Burch was named President-Elect and Dr. John L. Myers was elected Secretary.

XII

Chairman Babbitt reported that the annual business meeting had not been well attended.

Moved by Dr. Ralph I. Lloyd, seconded by Dr. Frank R. Spencer that the matter of the annual business meeting be referred to the Program Committee which will meet in January. Carried.

XIII

XIV

Program — 1944 Convention

Moved by Dr. C. H. McCaskey, seconded by Dr. Frank R. Spencer that the meeting in 1944 be increased to a five-day program. Carried.

XV

Moved by Dr. Frank R. Spencer, seconded by Dr. Albert C. Snell that a vote of thanks be given to the Board of Secretaries for the work they have done in preparing for this convention and especially to the Committee on the Revision of the Constitution and By-Laws. Carried.

The meeting adjourned.

Members who anticipate changes of address are asked to send them to the office of the Executive Secretary, 259 One Hundred First Avenue Building, Rochester, Minnesota, 30 days before the mailing date of the TRANSACTIONS in order to guarantee its delivery.

Business Meeting

OCTOBER 12, 1943

MINUTES

The business meeting of the American Academy of Ophthalmology and Otolaryngology, held in connection with its 1943 annual session, convened at five-thirty p.m. in the Crystal Room of the Palmer House, Chicago, Illinois, Dr. James A. Babbitt, President, presiding.

Secretary's Report

Secretary Benedict presented his report as follows:

SEPTEMBER 1, 1943

Fellows:		
Honorary Fellows		20
Active Fellows		
Life		248
Senior (dues paid)	181	
Senior (delinquent)	3	
	184	184
Junior (dues paid)	2,683	
Junior (delinquent)	189	
	2,872	2,872
Excused from payment of dues		11
		3,335
Summary of Fellowship:		
Active and Honorary Fellows Sept. 1, 1943	3,335	
Excused from paying dues, Council action, 1943	4	
Deferred action (delinquent), Council action, 1943	3	
	3,342	3,342
New Fellows, 1943		219
TOTAL FELLOWSHIP Oct. 12, 1943		3,561
Resignations, 1943	4	
Deaths, 1943	36	
Dropped for non-payment of dues, 1943 Council action	19	
(Deducted from above count of Active Fellows	59	
Summary of Candidates:		
Complete candidates, 1944	2	
Incomplete candidates	13	

Elected to Senior Fellowship, 1944:
Benedict, W. L., Rochester, Minn.
Blackburn, W. J., Dayton, Ohio
Boucher, R. B., Vancouver, B. C., Can.
Bribach, Eugene J., Atchison, Kansas
Burns, S. S., St. Louis, Missouri
Campbell, Duncan A., Detroit, Mich.
Campbell, W. K., Long Branch, N. J.
Cowper, H. W., Buffalo, New York
Cross, G. H., Chester, Pennsylvania
Curran, E. J., Kansas City, Missouri
Davis, William Thornwall, Washington, D. C.
Elles, Norma B., Houston, Texas
Farmer, A. G., Dayton, Ohio
Forsythe, S. T., Cleveland, Ohio
Gifford, S. R., Chicago, Ill.
Gill, E. G., Roanoke, Va.
Gordon, J. S., Milwaukee, Wis.
Harbert, John P., Bellefontaine, Ohio
Hill, C. E., Toronto, Ont., Can.
Hurff, J. Wallace, Newark, N. J.
Jones, F. W., Alton, Ill.
Krieger, William A., Poughkeepsie, N. Y.
Lunn, J. G., Pittsburgh, Pa.
Lubman, Max, New York, N. Y.
McDonald, C. L., Cleveland, Ohio
Macnie, J. S., Minneapolis, Minn.
Merrill, H. G., San Diego, Calif.
Miller, Alice R., West Palm Beach, Fla.
Moore, George H., Upper Darby, Pa.
Nelson, Charles F., Cleveland, Ohio
Novak, Frank J., Jr., Chicago, Ill.
Patterson, W. E., Minneapolis, Minn.
Prangen, Avery de H., Rochester, Minn.
Raydin, B. D., Evansville, Ind.
Sallander, F. W., Visalia, Calif.
Schwartz, Frederick O., St. Louis, Missouri
Shapero, I. M., Rochester, N. Y.
Shemeley, William G., Jr., Camden, N. J.
Sloan, Henry L., Charlotte, N. C.
Stimson, George W., Warren, Ohio
Tangeman, H. F., Cincinnati, Ohio
Vaughn, E. H., Tyler, Texas
Waugh, J. M., Willoughby, Ohio
Wolfe, Otis R., Marshalltown, Iowa
Zehnder, A. Charles, Newark, N. J.

Elected to Life Fellowship, 1944:
Bardenheier, F. G. A., St. Louis, Missouri
Beach, S. Judd, Portland, Maine
Blum, Henry N., New Orleans, Louisiana
Clark, J. Sheldon, Freeport, Ill.
Collins, J. D., Northampton, Mass.
Cross, A. E., Worcester, Mass.

Dow, Frank E., Northampton, Mass.
Dowling, J. Ivimey, Albany, N. Y.
Dutrow, H. V. (Major), U. S. Army
Flatley, T. J., Moline, Illinois
Grove, W. E., Milwaukee, Wisconsin
Hardy, W. E., St. Louis, Missouri
Inglis, Harry J., Boston, Mass.
McDowell, N. D., Rochester, New York
Marshall, Watson, Pittsburgh, Pa.
Messinger, H. C., Providence, R. I.
Muncy, William M., Providence, R. I.
Nutter, C. F., Nashua, New Hampshire
Parsons, J. G., Crookston, Minnesota
Ridpath, Robert F., Philadelphia, Pa.
Smith, George B., Rome, Georgia
Steim, J. M., New Kensington, Pa.
Thompson, Peter H., Boston, Mass.
Turner, H. H., Pittsburgh, Pa.
Weaver, T. Walker, Wichita, Kansas
Wise, Ralph C., Mansfield, Ohio
Wright, J. William, Indianapolis, Indiana

Upon motion made by Dr. Frank R. Spencer, seconded by Dr. Ralph I. Lloyd, it was voted that the report be accepted as read.

Secretary-Treasurer Benedict presented the report on Examination of Accounts for the year ended August 31, 1943, prepared by Shannon & Byers, Certified Public Accountants.

Treasurer's Report

SHANNON & BYERS
Certified Public Accountants
ROCHESTER, MINNESOTA

October 6, 1943

American Academy of
Ophthalmology and Otolaryngology
Rochester, Minnesota

Gentlemen:

As requested, we have examined the accounts and records of the American Academy of Ophthalmology and Otolaryngology, an unincorporated association, for the fiscal year ended August 31, 1943, and have prepared therefrom the accompanying Balance Sheet, Statement of Income and Expenses, and Analysis of Net Worth, together with schedules showing a summary of the Executive Secretary and Treasurer's cash transactions for the year and a list of securities owned on August 31, 1943. We have also reviewed the system of internal control and accounting procedures and, without making a detailed audit of cash receipts and disbursements and other transactions, have examined or tested accounting records and other supporting evidence, by methods and to the extent we deemed appropriate.

The amount of cash on deposit was verified by communicating with the various banks or through correspondence received from the banks by the Executive Secretary.

Investments in stocks and bonds had a market value of $94,424.46 on August 31, 1943, as shown in Schedule No. 2.

Securities in the Research Fund are held for safekeeping by the Trust Department of the Omaha National Bank, Omaha, Nebraska. U. S. Savings Bonds in the Wherry Memorial Fund are held by the Executive Secretary-Treasurer and were inspected by us. Interest earned since the dates on which the last coupons became due has not been accrued.

Furniture, fixtures, and equipment amounting to $1,038.01 represents equipment purchased during the past fiscal year and was for the most part verified through an inspection of supporting vouchers. Equipment acquired in prior years, which was charged to operating expenses as purchased, has not been set up on the balance sheet.

Current liabilities of $216.63 consist of federal Victory tax and Withholding tax deductions. The current general expenses are usually paid in the month following the one in which they are incurred, and those payable at the close of the fiscal year have therefore not been included under current liabilities. However, it was found that all employees' salaries due for the fiscal year had been paid by August 31, 1943.

Respectfully submitted,
SHANNON & BYERS,
Certified Public Accountants
By L. J. Byers, C.P.A.

Exhibit "A"

AMERICAN ACADEMY OF
OPHTHALMOLOGY AND OTOLARYNGOLOGY
Balance Sheet as at August 31, 1943

ASSETS

Current Assets:		
Cash, First Nat'l Bank, Rochester, Minn.	$21,965.95	
Cash, Bank of Montreal, Toronto, Ontario	1,553.71	
Cash, Omaha Nat'l Bk., Trust Dept., Omaha	1,286.00	
Total cash, (Schedule 1)	$24,805.66	
Foreign checks in transit	15.00	
Total current assets		$ 24,820.66
Investments (Schedule 2):		
Stocks and bonds (at market value)		94,424.46

Furniture, Fixtures and Equipment	1,038.01
Total Assets	$120,283.13

LIABILITIES

Current Liabilities:	
Accrued Federal Victory and Withholding taxes	$ 216.63
Net Worth (Exhibit "C")	120,066.50
Total Liabilities and Net Worth	$120,283.13

Exhibit "B"

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Statement of Income and Expenses

Year Ended August 31, 1943

Revenues:		
Membership dues		$18,650.00
Membership applications		3,808.00
Atlases		2,221.25
Transactions		14,981.90
Home Study Courses.....		1,725.00
Conventions		23,861.24
Contributions, Wherry Memorial Fund		4,660.00
Income from securities:		
Int., Research Fund...	$ 2,089.93	
Div., Research Fund..	1,091.08	
	$ 3,181.01	
Interest, Wherry Memorial Fund	50.00	3,231.01
Sundry receipts		566.53
Total revenues		$73,704.93
Operating Expenses:		
General administrative expenses	$17,253.62	
Transactions	16,575.27	
Home Study Courses	3,961.30	
Convention and meeting expenses	19,488.92	
Research	2,929.66	
Wherry Memorial Fund expenses	24.50	
Total Expenses		60,233.27
Revenues in excess of operating expenses		$13,471.66
Less: Loss on securities sold during year		4,681.90
Net Income		$ 8,789.76

Exhibit "C"

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Analysis of Net Worth Account

Year Ended August 31, 1943

Net worth, August 31, 1942 (Per audit report) (*)		$ 97,246.90
Additions during year:		
Net income, year ended Aug. 31, 1943 (per Exhibit "B")	$ 8,789.76	
Increase in market value of securities from Sept. 1, 1942, to Aug. 31, 1943 (†)	14,029.84	
		22,819.60
Net worth, August 31, 1943 (*) .		$120,066.50

(*) Does not include present market value of office furniture, fixtures, and equipment purchased prior to September 1, 1942, which had been charged directly to operating expenses.

(†)

	COST	MARKET	INCREASE (DECREASE)
8/31/42	$115,281.73	$82,225.33	($33,056.40)
8/31/43	113,451.02	94,424.46	(19,026.56)
Diff'rnce	$(1,830.71)	$12,199.13	$14,029.84

Schedule No 1

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Summary of Executive Secretary and Treasurer's Cash Transactions

For Year Ended August 31, 1943

Cash on hand, August 31, 1942 (Per audit report)		$15,021.57
Cash receipts by Treasurer.....		87,934.56
		102,956.13
Less: Check disbursements.....		79,436.47
Balance, August 31, 1943.....		$23,519.66
Add: Transactions by Omaha National Bank, Trust Dept., Omaha, Neb. (Custodian):		
Receipts from sale of securities, etc.	$17,775.16	
Less: Cost of securities purchased	16,489.16	
Balance, August 31, 1943.....		1,286.00
Cash on Hand, August 31, 1943 (Exhibit "A")		$24,805.66

SCHEDULE NO. 2

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Securities Owned, August 31, 1943

RESEARCH FUND

BONDS	RATE	MATURITY	PAR VALUE	COST	MARKET VALUE
American International Corp.	5½	1/1/49	$ 2,000.00	$ 1,962.50	$ 2,115.00
Brooklyn Edison Co., Inc.	3½	5/15/66	3,000.00	3,045.00	3,330.00
Canada, Dominion of—1931 Conv. Loan	4½	11/1/58	5,000.00	4,950.00	4,925.00
C. M. St. P. & P. R. R. Co.—Mtge. Ser. A.	5	2/1/75	2,000.00	1,821.50	740.00
Citizens Bldg. Co. of Cleveland 1st Mtge.	6	1/1/46	5,000.00	5,000.00	.....
Denmark, Kingdom of—External loan	5½	8/1/55	2,000.00	2,020.00	1,330.00
Kansas City, Ft. Scott & Memphis Ry. Co.—Ref. Mtge. (Cert. of deposit)	4	10/1/36	5,000.00	4,352.50	4,250.00
New York, City of—Corporate stock for transit unification	3	6/1/80	4,700.00	4,700.00	5,076.00
Union Pacific R. R. Co.—35 yr. Deb.	3½	5/1/71	6,000.00	5,947.50	6,670.00
U. S. Savings, Series D.	.....	4/1/49	10,000.00	7,500.00	8,200.00
U. S. Savings, Series D.	.....	3/1/50	10,000.00	7,500.00	8,100.00
U. S. Savings, Series G.	2½	8/1/53	10,000.00	10,000.00	9,620.00
U. S. Savings, Series G.	2½	6/1/55	4,000.00	4,000.00	3,952.00
Van Swerigen Co., Cert. of Indebtedness	6	12/31/48	6,000.00	6,031.33	480.00
Total Bonds				$68,830.33	$58,788.00

STOCKS	KIND	SHARES		
Citizens Bldg. Co. of Cleveland		50	None	.....
Commonwealth Edison Co.	Common	100	$ 2,619.00	$ 2,700.00
Du Pont de Nemours & Co.	Common	15	2,347.31	2,197.00
Louisville Gas & Electric Co.	5% Preferred	200	5,450.00	4,150.00
Massachusetts Investors Trust		978	27,673.90	20,117.46
Ophthalmic Publishing Co.	Capital	5	None	.....
Philip Morris & Co., Ltd.	Common	30	2,530.48	2,520.00
Total Stocks			$40,620.69	$31,684.46
Total		(A)	$109,451.02	$90,472.46
WHERRY MEMORIAL FUND				
U. S. Savings, Series G.		(B)	4,000.00	3,952.00
Total			$113,451.02	$94,424.46

(A) Held for safekeeping by Trust Dept. of Omaha National Bank, Omaha, Neb.
(B) Held by Dr. W. L. Benedict, Executive Secretary-Treasurer, Rochester, Minn.

Upon motion made by Dr. Frank R. Spencer, seconded by Dr. Ralph I. Lloyd, it was voted that the report be accepted and placed on file.

In presenting the Necrology report for 1943, Secretary Benedict stated that the names of the deceased Fellows would be published in the TRANSACTIONS in the minutes of the annual meeting. The names are as follows:

Barnhill, John Finch, Miami Beach, Fla.
Beck, Joseph C., Chicago, Ill.
Blakesley, Theodore S., Kansas City, Mo.
Day, Dwight Wilbur, Pittsburgh, Pa.
Duane, Joseph F., Peoria, Ill.
Fouts, John Daniel, Dayton, Ohio
Geiger, Arthur Henry, Chicago, Ill.
Greenwood, Allen, Boston, Mass.
Griscom, John Milton, Moorestown, N. J.
Harris, Will C., Cincinnati, Ohio
Hartley, Frank Allen, Springfield, Ohio
Ingerman, Surgius M., New York, N. Y.
Jackson, Edward, Denver, Colorado
Johnston, Wilson, Portland, Oregon
Lapsley, Robert McKee, Keokuk, Iowa
Loring, Jonathon Brown, Chicago, Ill.
McKee, S. Hanford, Montreal, Canada
Mengel, Willard Geist, Camden, N. J.
Muncaster, Stewart Brown, Washington, D.C.
Norwood, Eugene P., Corsicana, Texas
Oast, Samuel Poindexter, New York, N. Y.
Peter, Luther Crouse, Philadelphia, Pa.
Poos, Grover Hewitt, Palm Spring, Calif.
Robinson, Howard Thomas, Cumberland, Md.

Rosenthal, Joseph M., Philadelphia, Pa.
Schiller, Abraham Noah, New York, N. Y.
Sebert, Louis Joseph, Toronto, Canada
Sener, Walter J., Media, Pa.
Simon, Frederick Casimir, St. Louis, Mo.
Stokes, Harry Bayliss, Omaha, Nebraska
Strong, James Craig, Leadville, Colorado
Stroud, Edward Franklin, Corpus Christi, Texas
Vinsonhaler, Frank, Little Rock, Arkansas
Wagner, Carl B., Chicago, Ill.
Walker, C. B., Los Angeles, Calif.
Wherry, Walter Joseph, Omaha, Nebraska

Dr. Frank E. Brawley presented the report of the Senior Member of the Council, as follows:

DR. BRAWLEY: The Council has approved the revision of the Constitution. It is not necessary to read the Constitution at this time, but merely to present it to you. The Constitution will be printed in the TRANSACTIONS for your consideration and then will be presented to you for final action at the next annual meeting.*

The Council has also approved the revision of the By-Laws, and as these must be considered at this meeting, it will be necessary to read the By-Laws to you at this time.

Suggested Changes in the By-Laws

The Committee recommends that Article I of the By-Laws, which reads:

ANNUAL MEETING

The Annual Meeting shall convene at such time as the Council may recommend. The place of meeting shall be decided by the Council, notice of which shall be given to each member at least three months in advance.

Be amended to read as follows:

ANNUAL MEETING

The Annual Meeting shall convene at such time as the Council may recommend. The date and place of the meeting shall be decided by the Council, notice of which shall be published in the TRANSACTIONS at least three months in advance.

Article II of the By-Laws which reads:

PAPERS

Twenty minutes shall be the maximum time allowed for the reading of a paper. The Fellow designated to open the discussion shall be allowed ten minutes. In the general discussion, no one shall speak more than five minutes, nor shall he discuss any paper more than once. All such papers shall be edited by the Editor of the TRANSACTIONS.

No member shall read a paper that has been previously read or published.

All papers read before the Academy shall immediately become the property of the Academy, and must be delivered to the Secretary at the conclusion of their reading. All such papers shall be edited by the Editor of the TRANSACTIONS and their publication in the TRANSACTIONS shall be under the control of the Council.

Every paper must be read in person by its author, unless good and valid reasons for his absence can be shown, when, upon a unanimous vote of the Fellows present, the paper may be read by proxy.

Be amended to read as follows:

PAPERS

Twenty minutes shall be the allotted time allowed for the reading of a paper. The Fellow designated to open the discussion shall be allowed ten minutes. In the general discussion, no one shall speak more than five minutes, nor shall he discuss any paper more than once.

No Fellow shall read a paper that has previously been submitted elsewhere for publication.

All papers read before tthe Academy shall immediately become the property of the Academy and must be delivered to the Secretary in charge at the conclusion of their reading. All such papers shall be edited by the Editor of the TRANSACTIONS and their publication in the TRANSACTIONS shall be at the discretion of the Editorial Board.

Every paper must be read in person by its author unless good and valid reasons for his absence can be shown, when, upon a unanimous vote of the Fellows present, the paper may be read by proxy.

(The Committee recommends that Article III of the By-Laws be deleted from the By-Laws and incorporated in Article V of the Constitution.)

Article III of the By-Laws be as follows:

THE COUNCIL

In order to facilitate the work of the Council, Council committees shall be appointed annually by the President unless otherwise herein provided for.

The following Council committees shall be maintained:

* The revised Constitution is contained on Page 111.

1. *The Executive, Research and Finance Committee*

The Executive, Research and Finance Committee shall consist of the President, who shall be the Chairman, the three most recent Past Presidents, the President-Elect, the Senior of the elected Councillors and the Executive Secretary-Treasurer.

This Committee shall have charge of the affairs of the Academy when the Council is not in session and shall convene for transaction of business at the call of the chairman.

The following items of business shall be referred to the Executive, Research and Finance Committee which will in turn report recommendations to the Council.

(a) Entrance fee, annual dues and assessments.
(b) Disposition of Fellows delinquent in dues.
(c) Resignations and re-instatement of Fellows.
(d) Nomination of Honorary Fellows.
(e) Nomination to standing and joint committees.
(f) Nomination of Editor-in-Chief, TRANSACTIONS.
(g) Time and place of annual meeting.
(h) Matters involving current expenditures of funds, grants, and management of all Academy funds and property.
(i) Salaries and honorariums.

A subcommittee of this Committee to be known as the Research and Finance Committee, composed of the Senior Member of the Council, the President and the Executive Secretary-Treasurer, shall have charge of the financial affairs of the Academy. The Senior Member of the Council shall be the Chairman. The Chairman of the Research and Finance Committee and the Executive Secretary-Treasurer shall be the fiscal agents of the Academy. All deeds, transfers, conveyances, assignments and other instruments necessary to sell, convey or transfer any property, real or personal, of this Academy, shall be signed by the Chairman of the Research and Finance Committee and by the Executive Secretary-Treasurer as fiscal agents, and shall have the seal of the Academy affixed.

2. *The Activities Committee*

The Activities Committee shall consist of the four Fellows elected as Councillors, the President-Elect and the First Vice President, the Senior Councillor to be Chairman.

The following items of business shall be referred to the Activities Committee which will in turn report recommendations to the Council:

(a) Reports of standing and joint committees other than Council Committees.

3. *The Judicial Committee*

The Judicial Committee shall consist of the President, who shall be Chairman, and the four elected Councillors.

It shall investigate all charges against Fellows and report its findings to the Council.

4. *The Board of Councillors*

The four Active Fellows elected as Councillors according to the provisions of Article V of the Constitution shall constitute the Board of Councillors. The Senior Councillor shall be the Chairman of the Board of Councillors. It shall be the Nominating Committee for officers of the Academy, except those otherwise provided for. It shall be the Membership Committee and pass upon all applicants for Fellowship. It shall nominate to the Council a successor or substitute to fill the place of an elective officer who for any reason is unable during the current year to perform his duties as otherwise provided.

5. *The Board of Secretaries*

The Board of Secretaries shall consist of the Executive Secretary-Treasurer, who shall be Chairman, a Secretary for Ophthalmology, a Secretary for Otolaryngology, two Secretaries for Instruction, a Secretary for the Home Study Courses and a Secretary for Public Relations. It shall be the duty of the Board of Secretaries to prepare the scientific programs, the scientific and technical exhibits and the program for instruction courses, and to perform all duties necessary for conducting the annual convention. It shall prepare and conduct Home Study Courses. It shall investigate residencies, fellowships, internships and assistantships available to candidates who wish to qualify as Fellows of this Academy.

6. *The Program Committee*

The Program Committee shall consist of:
President
President-Elect
First Vice President
General Chairman of Arrangements
Chairman of Council Committee on Academy Activities
Board of Secretaries

This Committee shall meet in January of each year, and shall plan the activities of the annual convention.

Article IV of the By-Laws which reads:

DUTIES OF OFFICERS, ETC.

Section 1 — The President. The President shall preside at all meetings of the Academy and of the Council, and shall act as the executive officer of the Academy, and shall be ex-officio Chairman of the Council and Chairman of the Program Committee. He shall fill by appointment all vacancies occurring in the personnel of the committees.

Section 2 — The President-Elect. The President-Elect shall attend the meetings of the Council with power to vote, and shall assume the duties of President when this office is vacated, either by the expiration of the term of the President or for any other reason.

Section 3 — The Vice Presidents. The Vice Presidents, in order of seniority, in the absence of the President, shall perform the duties of the President.

Section 4 — The Comptroller. The Comptroller shall be the financial officer of the Academy, representing and responsible to the Council. He shall approve all budgets, supervise the work done in the Executive Secretary-Treasurer's office, sign all vouchers with the Finance Committee, all investments and shall, in general, be responsible for the expenditures of all monies. He will provide a suitable bond and be allowed an honorarium by the Council.

Section 5 — The Executive Secretary-Treasurer. The Executive Secretary-Treasurer will conduct the business affairs of the Academy, collect dues, draw, sign and send all vouchers for payment of indebtedness to the Comptroller and Finance Committee, shall arrange with a proper Trust Company for the safekeeping of all monies and investments belonging to the Academy. He shall so keep the books of this Academy that a monthly audit can be sent to each member of the Council and the Comptroller. He shall be held responsible for the work done by each of the subofficers. He shall have the privilege of nominating, subject to the approval of the Council, the different secretaries of sections. He will provide a suitable bond and be allowed an honorarium by the Council.

Section 6 — The Editor of the TRANSACTIONS shall receive all papers and reports read before the Academy with the discussion of the same, and from them, with the aid of the Secretary (and with the sanction of the Council) compile and publish the TRANSACTIONS.

Section 7 — The Board of Councillors. The Board of Councillors shall be the Board of Trustees of the Academy, and shall meet at the call of the President.

It shall be the Nominating Committee for Officers.

It shall be the Membership Committee and pass upon all applicants for Fellowship.

It shall consider all matters pertaining to the welfare of the Academy. It shall investigate all charges against members, and arbitrate and decide by a majority vote all professional controversies that may come before the Council, and its decisions on such questions shall be final. It shall appoint a successor or substitute to fill the place of an elective officer, who, for any reason, is unable, during the current year, to perform his duties.

Section 8 — The American Board for Ophthalmic Examinations. Three of the nine members of the American Board for Ophthalmic Examinations represent the Academy. According to the By-Laws of the Board, one member is elected each year to serve three years, beginning the first of January next following the meeting at which he is elected. No member of the Board may serve more than six years continuously. Members shall be chosen in the same manner as the presiding officers of the Society which they represent.

In accordance with this arrangement, one Fellow shall be elected each year, on nomination by the Council, to serve for three years as a member of the American Board for Ophthalmic Examinations.

Section 9 — The American Board of Otolaryngology. Two representatives of the Academy shall be elected every six years to serve as directors for the American Board of Otolaryngology.

Section 10 — The Program Committee. The Program Committee shall consist of:

President
President-Elect
First Vice President
General Chairman of Arrangements
Chairman of Council Committee on Academy Activities
Comptroller
Board of Secretaries

It shall also be known as the Committee on Arrangements. This group shall meet early in January of each year.

Section 11 — The Board of Secretaries. The Section Secretaries shall constitute the Board of Secretaries of which the Executive Secretary and Treasurer shall be chairman.

Section 12 — The Necrologist. The Necrologist shall be appointed by the President, and he shall present at the annual meeting a report of the recent death of any Fellow and prepare a brief biographic sketch of each deceased member.

Section 13 — The Research Committee. The Research Committee shall be a standing Committee of the Council and shall consist of the three most recent past presidents.

This Committee shall have the privilege of recommending to the Council for approval expenditures for research purposes from the interest accumulating from capital investments.

Be amended to read as follows:

DUTIES OF OFFICERS

Section 1 — The President. The President shall preside at all meetings of the Academy and shall be ex-officio Chairman of the Council and Chairman of the Program Committee. He shall fill by appointment all vacancies occurring in the personnel of the standing and joint committees not otherwise provided for.

Section 2 — The President-Elect. The President-Elect shall assume the duties of President when this office is vacated either by the expiration of the term of the President or for any other reason.

Section 3 — The Vice Presidents. The Vice Presidents, in order of seniority, in the absence of the President shall perform the duties of the President.

Section 4 — The Executive Secretary-Treasurer. The Executive Secretary-Treasurer shall act as the executive officer of the Academy, shall conduct the business affairs of the Academy, collect dues, draw, sign and send all vouchers for payment of indebtedness. He, with the approval of the Research and Finance Committee, shall arrange with a Bank and/or Trust Company for the safe custody of all monies and investments belonging to the Academy. He shall so keep the books of this Academy that a monthly statement can be sent to each member of the Council.

He shall keep a record of the Academy meetings, as well as of the Council and Program Committee meetings, of which he shall be ex-officio clerk. He shall be the Chairman of the Editorial Board and shall superintend the publication of the TRANSACTIONS.

He shall have the privilege of nominating, subject to the approval of the Council, the other Secretaries, and he shall be held responsible for the work done by each of them.

He shall provide a suitable bond and be allowed an honorarium.

Section 5 — The Editorial Board. The Editorial Board shall consist of an Editor-in-Chief, to be appointed by the Executive, Research and Finance Committee, an Associate Editor for Ophthalmology and an Associate Editor for Otolaryngology, to be appointed by the Editor-in-Chief, and the Board of Secretaries. The Executive Secretary-Treasurer shall be the Business Manager of the TRANSACTIONS. The Editorial Board will review and pass upon reports of completed research, monographs and other articles submitted for publication, and in conjunction with the Research and Finance Committee arrange for publication as supplements to the TRANSACTIONS such contributions as may be approved.

The Editor of the TRANSACTIONS. The Editor-in-Chief shall receive all papers and reports read before the Academy with the discussions of the same, and from them, with the aid of the Editorial Board and with the sanction of the Council, compile and publish the TRANSACTIONS.

Section 6 — The Necrologist. The Necrologist shall be appointed by the President. He shall prepare a brief biographical sketch of each deceased Fellow for publication in the TRANSACTIONS and report the names at the Executive Session.

Article V of the By-Laws which reads:

DUES

Section 1. The fiscal year of the Academy shall commence January first, and the current annual dues of ten dollars ($10.00) are payable at this time. The entrance fee paid by the newly elected Fellow shall cover his annual dues to December thirty-first following his election.

Section 2. A copy of the TRANSACTIONS of the preceding meeting shall be forwarded free of charge to all Fellows whose annual dues are paid for the year concerned.

Section 3. A member in arrears for two years' dues, who fails, after notification by the Secretary-Treasurer to last address, to pay such arrears, must be dropped from the roll.

Be amended to read as follows:

DUES

Section 1. The entrance fee and the annual dues and assessments shall be fixed yearly by the Council.

Section 2. The operating year shall commence on January 1 of each year and the current annual dues are payable at this time. The entrance fee paid by a newly elected Fellow shall cover his annual dues to December 31 following his election.

Section 3. A portion of the current annual dues paid by each Fellow, as determined by the Council, shall be designated as a subscription fee to the TRANSACTIONS which shall

be mailed to all Fellows whose annual dues are paid for the year concerned. The current TRANSACTIONS shall be mailed to Honorary and Life Fellows without charge.

Section 4. A Fellow in arrears for one year's dues, who fails after notification by the Executive Secretary-Treasurer to last known address to pay such arrears, must be dropped from the subscription roll of the TRANSACTIONS.

Section 5. A Fellow in arrears for three years' dues, who fails after notification by the Executive Secretary-Treasurer to last known address to pay such arrears, shall be reported to the Council as delinquent. The Council will then decide whether such delinquent Fellow shall be dropped from the Fellowship roll of the Academy.

Section 6. Fellows shall be classified as follows:

Honorary Fellows. Honorary Fellows shall not pay dues. They shall not be eligible to hold office nor to vote.

Active Fellows shall be classified as follows:

Life Fellows. Fellows who have paid dues continuously for thirty years will automatically become Life Fellows. They shall not be required to pay any fees or dues or assessments and shall enjoy all the privileges of Active Fellows.

Senior Fellows: Fellows who have paid dues continuously for twenty-five years will be termed Senior Fellows and their dues will be one-half of the dues of Junior Fellows.

Junior Fellows: All other Fellows will be designated as Junior Fellows. Dues will be determined yearly by the Council.

The Council, for sufficient reasons, may nominate Fellows to Life or to Honorary Fellowship, and a unanimous vote of the Active Fellows present and voting at the annual meeting shall constitute an election.

Article VI of the By-Laws which reads:

RE-ELECTION and RE-INSTATEMENT

Section 1. A Fellow whose name has been dropped from the roll for non-payment of dues, as provided in Article V of the By-Laws, may be re-admitted to Fellowship by formally applying for admission as a new member.

Section 2. A Fellow who resigns from Fellowship in the Academy may be re-instated at the direction of the Council by conforming to the requirements of the Society, as provided in Article III of the constitution.

Be amended to read as follows:

RE-ELECTION and RE-INSTATEMENT

Section 1. A Fellow whose name has been dropped from the Fellowship roll of the Academy for non-payment of dues, as provided in Article V of the By-Laws, may be re-admitted to Active Fellowship only by fulfilling the requirements for Fellowship for admission as a new Fellow.

Section 2. A Fellow in good standing who resigns from Fellowship in the Academy may be re-instated at the direction of the Council by conforming to the requirements of the Society, as provided in Article III of the Constitution.

Article VII of the By-Laws which reads:

BUSINESS SESSION

The regular Business Session shall be held some evening during the latter part of the annual convention,

and

Article VIII of the By-Laws which reads:

ORDER OF BUSINESS

1. Call to order.
2. Report of Executive Secretary and Treasurer.
3. Report of Necrologist.
4. Report of Editor of TRANSACTIONS.
5. Report of Senior Member of Council.
6. Committee reports.
7. Report of American Board for Ophthalmic Examinations.
8. Report of American Board of Otolaryngology.
9. Unfinished business.
10. New business.
11. Election of members.
12. Election of officers.
13. Place of meeting.

Be amended to read as follows:

ARTICLE VII
ANNUAL BUSINESS MEETING

An Annual Business meeting shall be held during the annual convention at a date and place to be determined by the Council.

Order of business:

1. Call to order.
2. Report of Executive Secretary-Treasurer.
3. Report of Necrologist.
4. Report of Editor of TRANSACTIONS.
5. Committee reports.
6. Report of American Board of Ophthalmology.
7. Report of American Board of Otolaryngology.

8. Unfinished business.
9. New business.
10. Election of Fellows.
11. Report of the Senior Member of the Council.
12. Election of officers (Senior Member of Council report).

DR. BRAWLEY: Is it your desire that the By-Laws be adopted as a whole, or do you wish them read again and adopted section by section?

DR. THOMAS E. CARMODY: I move that they be acted on as a whole.

DR. JAMES M. ROBB: I second the motion.

PRESIDENT BABBITT: A motion is made and seconded that these By-Laws be acted on as a whole. Of course, the Constitution has to be published and acted upon at our next annual meeting, but we are privileged to vote upon the By-Laws now. The Committee on the Revision of the Constitution and By-Laws has worked very faithfully. Are you ready for the question?

The motion was put to a vote and carried.

DR. BRAWLEY: Reports from the various Standing and Joint Committees have been given to the Council. These reports have been read and considered by the Activities Committee. There are no significant items that would require any change in our policies. I should like to dispense with the reading of all these with your consent, as they will be published in the TRANSACTIONS.

DR. BURT R. SHURLY: I move that the reading of the Committee reports be dispensed with.

DR. LAWRENCE T. POST: I second the motion.

PRESIDENT BABBITT: It has been moved by Doctor Shurly and seconded by Doctor Post that the reading of the reports of the Standing and Joint Committees be dispensed with.

The motion was put to a vote and carried.

STANDING COMMITTEES

AMERICAN BOARD OF OPHTHALMOLOGY

September 1, 1943

The American Board of Ophthalmology submits the following report:

Since the last meeting of the Academy, examinations were conducted in New York in December, 1942, in Los Angeles in January, 1943, and in New York in June, 1943. To date 81 examinations have been held and a total of 2,222 certificates have been issued.

Written examinations and case reports have been dispensed with for the duration, and the preparatory group has been discontinued.

At the last Board meeting it was voted not to reduce requirements and not to lower standards. The subjects previously covered in the written examination will now be included in Part I examination, which will be followed by the clinical examination (Part II). At least two days of examinations are required of each candidate.

Because of the large number of candidates and the war emergency, the Board announces the following additional examinations:

Chicago, October 8 and 9, 1943

The Board also decided not to reduce the minimum time requirements in the specialty, viz.: three years in ophthalmology and five years in otolaryngology combined with ophthalmology.

Forty ophthalmologists have served on the Board since its organization in 1917.

Beginning January 1, 1944, the fee for the examination will be $75.

The 1943 personnel of the Board is as follows:

A. O. S.	*Sec. on Oph.*	*Academy*
Term Ends—December 31, 1943		
W. Lancaster	T. L. Terry	F. C. Cordes
Term Ends—December 31, 1944		
John Green	S. J. Beach	W. Benedict
Term Ends—December 31, 1945		
G. Theobald	D. B. Kirby	C. S. O'Brien
Term Ends—December 31, 1946		
E. C. Ellett	C. Berens	A. B. Reese
Term Ends—December 31, 1947		
M. H. Post		

FREDERICK C. CORDES, M.D.

AMERICAN BOARD OF OTOLARYNGOLOGY

September 1, 1943

The American Board of Otolaryngology conducted an examination in Chicago in October, 1942. One hundred-thirty-nine candidates were examined of whom 103 passed. At the time of the New York examination in June, 1943, 150 candidates were examined of whom 113 passed.

DEAN M. LIERLE, M.D., *Sec'y*

REGISTRY OF OPHTHALMIC PATHOLOGY

September 23, 1943

Advisory Committee

Dr. Harry S. Gradle, Chicago, Ill., *Chairman*
Dr. F. H. Verhoeff, Boston, Mass.
Dr. Jonas S. Friedenwald, Baltimore, Md.
Dr. Georgiana D. Theobald, Chicago, Ill.
Col. J. E. Ash, Curator, Army Medical Museum (*Ex-Off.*)

TABLE *I*

Year	*No. of Accessions*
1922-1934	4,847
1935	415
1936	570
1937	863
1938	972
1939	1,167
1940	1,441
1941	1,353
1942	1,488
	13,116

There were 1,488 accessions during the year 1942, the greatest number in the history of the Registry. The number of enucleated eyes received each month has increased considerably.

For the sixth year the course in ophthalmic pathology based on Registry material was given in connection with the Post-graduate Course in Ophthalmology at George Washington University.

Loan sets have again been in great demand, both by applicants for the certificate of the American Board of Ophthalmology and by individual doctors, some of whom have come to the Museum for study and special instruction in eye pathology. The Atlas, too, has been very popular and a revision and new edition are planned in the near future.

At the Academy meeting in October, 1942, an exhibit was prepared and shown on ophthalmic pathology from military personnel under the title The Registry at War.

J. E. ASH, Col., M. C., U. S. A., *Registrar*
HARRY S. GRADE, M.D., *Chairman*

REGISTRY OF OTOLARYNIC PATHOLOGY

September 23, 1943

Advisory Committee

Dr. Frank J. Novak, Chicago, Ill., *Chairman*
Dr. Gregor W. McGregor, Toronto, Canada
Dr. Herman Z. Semenov, Los Anegeles, Calif.
Dr. Werner Mueller, Boston, Mass.
Dr. Bernard J. McMahon, St. Louis, Mo.
Col. J. E. Ash, Curator, Army Medical Museum (*Ex-Off.*)

There were 226 accessions during the past year, a slight increase over the previous number, making a total of 2,054 cases in the Registry on June 30, 1943.

The loan sets have been extensively used and again we have had to carry a waiting list during the periods before the Board examinations. The Atlas, too, has been very popular and a revision and new edition are planned for the near future.

At the Academy meeting in October, 1942, an exhibit was prepared and shown on otolaryngic pathology from military personnel under the title The Registry at War.

J. E. ASH, Col., M. C., U. S. A., *Registrar*
FRANK J. NOVAK, M.D., *Chairman*

WAR ACTIVITIES

September 1, 1943

The War Activities Committee has found great difficulty in functioning in the face of the objections of the Surgeon General to all specialism. The unfortunate experiences of the last war, when some specialists endeavored to control and direct the Surgeon General's office in the A. E. F. and take over hospitals and supplies, were not for the general good of the Service. A complete lack of understanding of military courtesy and administration led to much trouble. Our contact with the Surgeon General has been courteous and pleasant, but full of complete resistance.

In this war the general surgeon commands the specialist, and the newer ruling places him in charge of orders and directions for the otolaryngologist and other specialists except psychiatrists and internists. Older men or those who served in the last war are not desired, and younger men of low rank are ordered into Service even to the complete depletion of residents of special hospitals.

The National Research Council has discharged the Sub-Committee on Otolaryngology although this committee produced a splendid manual under the direction of Dr. H. P. Mosher. Many valuable suggestions in regard to audiometers and the prevention of deafness were offered, and several research problems on the loss of hearing and hearing aids were presented. The discharge of this committee was protested without avail and the general surgeon again takes command of research problems in otolaryngology.

A conference with Surgeon General Magee, who would receive only the chairman, was quickly referred to Colonels Rankin and Hillman.

The following 15 points were submitted, most of which were discarded as unimportant or unworthy of adoption:

1. Request that a chief consultant in otolaryngology and a consultant in ophthalmology and otolaryngology be appointed in each Corps area.

2. That these consultants inspect the equipment in station and general hospitals. At the last examination of the American Board of Otolaryngology, of the candidates who were already in service one-half stated that their special equipment was satisfactory and one-half stated that it was antiquated. The equipment in surgery was evaluated by Doctor Mixter, and that of plastic surgery by Doctor Ivey. No one was asked to review the equipment for ophthalmology or otolaryngology until recently.

3. Under the present set-up, permission to operate is necessary from the head of the Surgical Department. This implies universality of knowledge which no general surgeon possesses, except in rare cases.

4. The Sub-Committee on Otolaryngology of the National Research Council was dissolved because it was too large, and it was said that the Surgeon General did not feel the need of it. The Sub-Committee on Ophthalmology was retained. It is requested that a small committee be re-established in otolaryngology.

5. Our Academy Committee is keenly interested to know how much use is being made of the audiometer.

6. In the last three years, 46 Army medical officers have been examined and certificated by the American Board of Otolaryngology. We would like to have an increase of certification in this Service.

7. Many young men who are teaching in the medical schools are very patriotic and feel that unless some recognition from the Armed forces is given for their services, they should enlist and leave their teaching positions for those in which they are essential. Is there some way in which these men could be inducted into the Armed Forces and then assigned to the various hospitals for teaching medical students, at the same time having them available for active duty in an emergency?

8. If a few post-graduate centers could be established, men who are in need of additional instruction could be accommodated; this could be accomplished at three or four large centers.

9. Too many men are inducted who have chronic suppurative otitis media. This has apparently resulted because of carelessness on the part of the medical examiner at the time of the initial examination. We feel that radical mastoid operations should be eliminated. A routine radiographic examination to detect cholesteatoma should be given. With careful local treatment men could be returned to limited duty.

10. Many resections of deflected septa and even tonsillectomies could be done at station hospitals which are now done at general hospitals, entailing loss of time and travel expense.

11. Qualified Corps consultants could accomplish much in eliminating inequalities and incompetence in medical care.

12. The libraries of medical schools and special societies should be made available to men in their neighborhood.

13. Young men in many posts need special supervision; too many radical operations are now performed.

14. Dr. Frederick Hill has made an excellent suggestion — that enlisted men in the Medical Department be given fundamental training for possibly three months in our class A hospitals, thus allowing the nurses to function almost exclusively in a supervisory capacity, which would help in the matter of shortage of nurses.

15. We believe that it is necessary that the residents in the various specialties and those essential in teaching be given every inducement to remain at their work until their job is finished, or others are secured to take their places.

Our contacts and correspondence with the Surgeon General of the Navy were received with courtesy, interest and patience. We discussed the 15 points submitted to the Army. Admiral McIntire was firm in saying that more men would be requested to take the Board examinations and efforts would be made to establish post-graduate courses as soon as a sufficient number of specialists could be spared and assigned to instruction. A plan was requested and it was sent to his office with a curriculum and outline of study requiring four weeks. This was submitted six months ago. An answer stated that the plan would be submitted to an advisory committee which would request consultation. It may have been lost in the innumerable pigeon holes of the department.

The army has felt that post-graduate instruction was not necessary in ophthalmology and otolaryngology as well as in all other specialties for reasons stated that your chairman was unable to understand. It was said that these men were so well prepared they did not need post-graduate work. The general surgical department of the Army, that has charge of the surgical supplies for eye, ear, nose and throat practice, admits an insufficient number of special instruments in various stations. Colonel Canfield of New Haven was assigned to the job at home and Colonel Vail of Cincinnati was assigned to the British Isles. The use of audiometers and hearing aids was discussed at length with the National Research Council. These items were considered to be of limited use in great centers only. At the time of our last report 384 types of audiometers had been supplied. A new hearing aid at a cost of $26.00 will be on the market soon. This should be supplied to the deafened by the U. S. Army just as artificial eyes and legs are supplied.

Interesting sessions were attended in the form of meetings of the consultants for audiometers and hearing aids and with the Advisory Board of Medical Specialties, Colonel Fitts of the Army and a Commander from the Navy. Promises were made that certificated men would be assigned to their specialty as far as is possible.

The offices of the Surgeons General requested confidential information from the American Board of Otolaryngology in regard to men who had failed or who had applied and were not examined. The Otolaryngological Board felt that it was unfair to give out confidential information which they had received until the applicant had had a chance to make good.

The Medical Department in all Armed Services has acquitted itself with glory, efficiency and loyalty. This certainly applies to the eye, ear, nose and throat specialists.

BURT R. SHURLY, M.D., *Chairman*

CONSERVATION OF HEARING

September 1, 1943

The Executive Committee of the Committee on the Conservation of Hearing held a special meeting on January 3, 1943. The committee has been re-organized and the membership now is as follows:

*DEAN M. LIERLE, M.D., *Chairman*
*ERNEST M. SEYDELL, M.D., *Secretary*
*James A. Babbitt, M.D.
*Gordon Berry, M.D.
Thomas E. Carmody, M.D.
†Howard Carter, B. S.
George M. Coates, M.D.
*Warren H. Gardner, Ph.D.
*William E. Grove, M.D.
†Stacy Guild, Ph.D.
Frederick T. Hill, M.D.
*Walter Hughson, M.D.
*Marvin F. Jones, M.D.
Charles E. Kinney, M.D.
*Carl H. McCaskey, M.D.
C. Stewart Nash, M.D.
*Horace Newhart, M.D.
Charles T. Porter, M.D.
Arthur W. Proetz, M.D.
†Scott N. Reger, Ph.D.
H. Marshall Taylor, M.D.
†Harold Westlake, Ph.D.
Fletcher D. Woodward, M.D.
Burt R. Shurly, M.D.

Preliminary studies reveal that there are many problems for investigation by the Committee on the Conservation of Hearing, some of which are:

1. To promote active interest in the conservation of hearing.
   - A. Special studies of specific otological diseases, such as otosclerosis, syphilis, congenital deafness, tuberculosis, acoustic neuritis, hyperplastic otitis media and acute and chronic suppuration of the middle ear tract.
   - B. Testing of school children (formulation of state programs to promote its adoption by encouraging sound state legislation).
     1. The use of audiometric testing in the prevention, early discovery and treatment of hearing deficiencies, and in the educational adjustment of the acoustically handicapped.
   - C. Testing of adults
     1. The use of audiometric testing and of routine health examinations in industry, in the prevention, early discovery and treatment of hearing deficiencies, and in the educational and vocational adjustment of acoustically handicapped personnel.
     2. Testing of individuals who have served in the Armed Forces with special reference to treatment, rehabilitation, et cetera.

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* Executive Committee
† Consultants

D. To endorse and support the work of the Council on Physical Therapy, especially with regard to:
   1. Standardization of instruments for hearing acuity, hearing aids and other acoustical apparatus.
   2. Ethical standards of manufacturers and salesmen in the sale of hearing aids to the public.

E. Education of physicians, educators, legislators and the general public regarding the problems of the hard of hearing.

F. Encouragement of research in regard to the problems of the hard of hearing.

G. Investigation and reorganization of undergraduate and post-graduate education in otology.

There are three objectives that the Committee is actively working on at the present time:

1. To promote active interest in the conservation of hearing by establishing a national program for the testing of school children with emphasis on follow-up medical treatment.
   a. State programs for testing of school children and model bills for state legislature are being prepared.
   b. A detailed syllabus giving a definite plan of procedure for the testing of school children is being formulated.
2. Rehabilitation of individuals in the Armed Forces with defective hearing is being planned in cooperation with the Veterans' Administration.
3. Appointment of state committees to promote the otological program proposed by the Committee.
   1. It was recommended, in order to facilitate the extensive program, that the general Committee be increased to include, if possible, two members of the Academy from each state. A Sub-Committee composed of Drs. Newhart, Chairman, Babbitt and Grove was appointed to select state representatives.
   2. The members suggested are as follows:

LIST OF NOMINEES FOR STATE COMMITTEES ON THE CONSERVATION OF HEARING AS OF AUGUST 8, 1943

Alabama

Harvey B. Searcy, M.D., 2501 Sixth St., Tuscaloosa
Ernist C. Pope, 2021 Sixth Ave. N., Birmingham

Arizona

Earl H. Brown, 130 S. Scott St., Tuscon
Mayo Robb, Professional Bldg., Pheonix

Arkansas

Col. Ralph H. Goldwaite, Army and Navy General Hospital, Hot Springs
Kingsley W. Cosgrove, Little Rock

California

J. M. Brown, 1136 W. 6th, Los Angeles
William H. Johnson, 1515 State St., Santa Barbara

Colorado

Thomas E. Carmody, 227 16th St., Denver
Frank R. Spencer, 2111 14th St., Boulder

Connecticut

Edward Whalen, 750 Main St., Hartford
Norton Canfield, 789 Howard Ave., New Haven

Delaware

William O. LaMotte, Med. Arts Bldg., Wilmington
William M. Pierson, Med. Arts Bldg., Wilmington

District of Columbia

George B. Trible, 1160 Connecticut Ave., N. W., Washington, D. C.
Richard W. Wilkinson, 1408 I. St., N. W., Washington, D. C.

Florida

H. Marshall Taylor, 111 W. Adams St., Jacksonville
W. T. Hotchkiss, 541 Lincoln Road, Miami Beach

Georgia

Murdock S. Equen, 144 Ponce de Leon Ave., N. E., Atlanta
William L. McDougall, Doctors' Building, Atlanta

Idaho

Casper W. Pond, Carlson Bldg., Pocatello
Wallace Bond, Twin Falls

Illinois

Walter H. Theobald, 307 N. Michigan Ave., Chicago
Thomas C. Galloway, 636 Church St., Evanston

Indiana

C. H. McCaskey, 20 N. Meridian St., Indianapolis
J. K. Leasure, 23 E. Ohio St., Indianapolis

Iowa

Dean M. Lierle, University Hospital, Iowa City
Gordon F. Harkness, Putnam Bldg., Davenport

Kansas

Ernest M. Seydell, 1st Nat'l Bank Bldg., Wichita
Laverne B. Spake, 750 Minnesota Ave., Kansas City

Kentucky

Joseph D. Heitger, Heyburn Bldg., Louisville
A. L. Bass, Heyburn Bldg., Louisville

Louisiana

F. E. LeJeune, 921 Canal St., New Orleans
W. A. Wagner, 200 Carondelet, New Orleans

Maine

Frederick T. Hill, 177 Main St., Waterville
Henry P. Johnson, 32 Dearing St., Portland

Maryland

Massachusetts

Gordon Berry, 36 Pleasant St., Worcester
Werner Mueller, 512 Beacon St., Boston

Michigan

Burt R. Shurly, 62 W. Adams St., Detroit
J. M. Robb, 1553 Woodward Ave., Detroit

Minnesota

Lawrence R. Boies, Med. Arts Bldg., Minneapolis
Horace Newhart, Med. Arts Bldg., Minneapolis

Mississippi

Robin Harris, 964 N. State St., Jackson
B. S. Guyton, 512 Van Buren Ave., Oxford

Missouri

Arthur W. Proetz, Beaumont Bldg., St. Louis
John L. Myers, Shukert Bldg., Kansas City

Montana

F. D. Hurd, 503 First Ave., N., Great Falls
Roy V. Marledge, Hart-Albin Bldg., Billings

Nebraska

L. H. Heine, 635 N. Main St., Fremont
J. Calvin Davis, 1615 Howard St., Omaha

Nevada

Earle L. Creveling, 17 N. Virginia St., Reno
John A. Fuller, First Nat'l Bank Bldg., Reno

New Hampshire

Raymond H. Marcotte, 196 Main St., Nashua
John A. Murtagh, Hitchcock Clinic, Hanover

New Jersey

C. C. Charlton, 124 S. Illinois Ave., Atlantic City
Oram R. Kline, 514 Cooper St., Camden

New Mexico

George W. Griswold, 211 W. Third St., Roswell
Harrison L. Bremer, First Nat'l Bank Bldg., Albuquerque

New York

C. Stewart Nash, 277 Alexander St., Rochester
Marvin F. Jones, 121 E. 60th St., New York City

North Carolina

Fred E. Motley, 106 W. 7th St., Charlotte
W. W. Eagle, Duke Hospital, Durham

North Dakota

Nelson A. Youngs, Grand Forks Clinic, Grand Forks
Henry P. Rosenberger, Bismarck

Ohio

Charles E. Kinney, 10515 Carnegie Ave., Cleveland
Russell G. Means, 80 S. 3rd St., Columbus

Oklahoma

L. Chester McHenry, Med. Arts Bldg., Oklahoma City
Theodore G. Wails, Med. Arts Bldg., Oklahoma City

Oregon

Frank B. Kistner, 1130 S. W. Morrison, Portland
Guy Lee Boyden, 1735 W. Wheeler, Portland

Pennsylvania

James A. Babbitt, 1912 Spruce St., Philadelphia
George M. Coates, 1721 Pine St., Philadelphia

Rhode Island

Frank W. Adams, 122 Waterman St., Providence
Frank C. MacCardell, 193 Waterman St., Providence

South Carolina

Walter J. Bristow, 1517 Hampton St., Columbia
Clay W. Evatt, 112 Rutledge Ave., Charleston

South Dakota

John B. Gregg, Security Nat'l Bank Bldg, Sioux Falls
Paul G. Bunker, Newberry Building, Aberdeen

Tennessee

John J. Shea, 1018 Madison Ave., Memphis
Charles Blassingame, 899 Madison Ave., Memphis

Texas

Claude Cody, 1304 Walker Ave., Houston
John D. Singleton, 1719 Pacific Ave., Dallas

Utah

L. F. Stauffer, Medical Arts Building, Salt Lake City
Vivian P. White, Tribune-Telegraph Building, Salt Lake City

Vermont

Erasmus G. Twitchell, 266 Main St., Burlington
George G. Marshall, Rutland

Virginia

F. D. Woodward, 103 E. Market St., Charlottesville
E. T. Gatewood, 501 E. Franklin St., Richmond

Washington

West Virginia

Mathew F. Zuback, Citizens Mutual Trust Bldg., Wheeling
J. Halleck Moore, 1050 Fifth Avenue, Huntington

Wisconsin

W. E. Grove, Wells Bldg., Milwaukee
A. E. Rector, Zuelke Building, Appleton

Wyoming

The film "The Right to Hear" which was produced under the direction of the Department of Otolaryngology and Oral Surgery, State University of Iowa College of Medicine, with the cooperation of the Department of Speech, was previewed by the Committee and several members of the Council. The movie has been shown to various state and county medical societies, parent-teachers associations, etc., as propaganda for the testing of school children. Four copies have been made, one for the Academy and three for extensive use in the state. The cost of the film was $420.62.

A meeting of the Executive Committee was held on June 6, 1943, in New York for the purpose of reviewing and discussing the papers to be presented at the Conservation of Hearing program in Chicago, October 10, 1943, during the Academy convention.

The following program was outlined:

1. What Can Be Accomplished by Conventional Methods of Treatment.
   Frederick T. Hill, M.D.
2. Recent Developments in Non-Surgical Therapy.
   LeRoy M. Polvogt, M.D. (by invitation)
3. Surgical Treatment of Impaired Hearing (Results Obtained by Using the Lempert-Endaural Fenestration Technique).
   Marvin F. Jones, M.D.
4. Use of Hearing Aids.
   Walter Hughson, M.D.
   Scott N. Reger, Ph.D.
5. Movie — "The Right to Hear."

At the meeting held on June 6, 1943, Sub-Committees were appointed as follows:

1. Publicity — Dr. Lierle, Chairman; Drs. Babbitt, McCaskey, Carter and Gardner.
2. Rehabilitation — Dr. Grove, Chairman; Drs. Berry, Hughson and Westlake.
3. Standardization of Terminology: Dr. Newhart, Chairman; Drs. Hill, Jones and Coates.

DEAN M. LIERLE, M.D., *Chairman*

AMERICAN ASSOCIATION FOR THE ADVANCEMENT OF SCIENCE

September 15, 1943

During the past years there have been no activities to report except for the passage of a resolution condemning the Wagner-Murray Senate Bill No. 1161.

H. S. GRADLE, M.D.

HOME STUDY COURSES

September 15, 1943

The Home Study Courses for 1942-1943 were beset with the many difficulties incident to the medical manpower situation. Consequently rigid rules and regulations were overridden and the endeavor was made to supply the men in Service according to their requests. Note in the following tabulation the number of Residents who were unable to complete their papers for correction, probably due to their entering the Armed Forces.

OPHTHALMOLOGY

Residents		
Number who completed the course	26	
Number incomplete	24	
		50
Number on Uncorrected List		106
Number withdrawn		6
Address unknown		1
Number of Registrants		163

OTOLARYNGOLOGY

Residents		
Number who completed the course	17	
Number incomplete	33	
		50
Number on Uncorrected List		23
Number withdrawn		6
Address unknown		2
Number of Registrants		81

Two incidents were particularly gratifying. Lieutenant-Colonel Derrick Vail, our Ophthalmic Consultant in England, organized the oculists on duty there and gave them correspondence work, based upon the Home Study material. This was all sent to him with the aid of the Surgeon General, who forwarded the bulky papers in the Army air mail pouch.

Similarly, Captain Greenberg organized the eye men on duty in Camp Crowder, Missouri, and gave them a shorter course, based upon the Home Study material that was sent to him.

Some of the Registrants are sending their papers for correction from Australia, from England, and from some of the other theatres of war. Under these circumstances timing of the course is entirely disregarded.

The Home Study Courses for 1943-1944 started on September 1, with a registration of,

Ophthalmology 98
Otolaryngology 58

The courses will be conducted as in the past and no essential changes are planned for the coming year.

THE COMMITTEE

STANDARDIZATION OF TONOMETERS

August 30, 1943

I. *Membership of the Committee.* Of the originally appointed members, Doctors Posner and Harrington were unable to serve actively because they were in the Armed Forces. Dr. Mark J. Schoenberg was added to the Committee.

II. *Nature of the Problem.* The standardization of tonometers involved two distinct problems (a) Checking tonometers now in the hands of clinicians in order to determine for their owners whether or not they are capable of giving reliable measurements; and (b) arrangements with manufacturers as to mutually acceptable standards of construction such that different tonometers of the same and of different manufacture can be expected, when new and in good mechanical condition, to give concordant and reliable results on the same patients.

III. *Other Agencies Engaged in This Same Field.* Prior to the establishment of our Committee extremely important and fruitful work had already been begun in this field by a Committee of the National Society for the Prevention of Blindness under the leadership of Doctors Schoenberg and Posner. This Committee has established a testing station in New York City to which tonometers can be sent for testing and calibration. Many tonometers have already been carefully measured by this station and their physical characteristics recorded. Several reports on the results of these tests have been published by Doctors Schoenberg and Posner. These reports have established the fact that many of the instruments alleged to be Schiotz tonometers differ so markedly from the original specifications of Schiotz that their use in connection with the Schiotz scale leads to errors of as much as 30-40%.

In addition this Committee of the National Society for the Prevention of Blindness has arranged with the Electrical Testing Laboratories, Inc., to test Schiotz type tonometers submitted to them by manufacturers in order to establish how closely these newly made instruments conform to the specifications of Schiotz. The Electrical Testing Laboratories, Inc., is a commercial corporation engaged in testing objects and instruments submitted to it for test by various manufacturers. It will test tonometers and report the results of its measurements and certify those which are acceptable at a small cost per instrument, the cost to be born by the manufacturer. One manufacturer has already arranged to submit his product to this independent testing station before marketing.

IV. *The Work of the Committee on Standardization of Tonometers.* In view of the excellent and fruitful work already started under the auspices of the National Society for the Prevention of Blindness your Committee has decided to cooperate intimately with that Committee in every way possible. The problems to be faced appear to be the following:

(1) Establishment of additional testing stations for old tonometers. Since the testing station established in New York by the National Society for the Prevention of Blindness can conveniently serve only a small part of the country, it would appear desirable to establish similar testing stations in other parts of the country. Your Committee has already established such a station at the Illinois Eye and Ear Infirmary under the direction of Doctor Kronfeld. It is proposed that the measurements to be made at this station will follow the same plan as those in New York under Doctor Schoenberg and the results of the two stations will be closely correlated. It is expected that Doctor Kronfeld will be able to submit a preliminary report of the work of his station at the time of the next meeting of the Academy. As the work of this second station develops your Committee will consider the establishment of other stations in other parts of the country. Up to the present time no funds have been required for the establishment of this new testing station, but in the further expansion of this work some financial support may be required. Your Committee respectfully requests that the Council give this matter its consideration.

(2) Many of the old tonometers submitted for testing at the New York and Chicago testing stations have been found to require cleaning, adjustment and minor repairs. It would be most desirable if arrangements could be made for the establishment of some commercially operated repair shop to which the owners of these defective tonometers

could be referred. No steps in this direction have as yet been undertaken.

(3) In relation to new tonometers of current manufacture your Committee is attempting to work out in collaboration with the manufacturers acceptable standards of construction. This involves questions of standard design of the instruments and questions of standards of tolerance of deviation from standard design. As noted above, the Electrical Testing Laboratories, Inc., are willing to measure and report on the precise physical characteristics of the instruments submitted to them for testing, but the responsibility devolves on the profession to determine what are the tolerable limits of variatoin in tonometer construction which are still compatable with acceptable clinical performance.

Your Committee does not believe that such standards of limits for acceptable variation should be adopted dogmatically, but rather believes these should be developed by mutual agreement arrived at by discussion between your Committee and the various manufacturers. Negotiations to this end have already been begun with several manufacturers all of whom have been found to be most cooperative, and one manufacturer has already arranged to submit his product both to the Electrical Testing Laboratories, Inc., for physical standardization and to your Committee for clinical evaluation. It is hoped that your Committee will shortly be able to report on the acceptability of this instrument.

JONAS S. FRIEDENWALD, M.D., *Chmn.*
FRANCIS HEED ADLER, M.D.
SANFORD R. GIFFORD, M.D.
HARRY S. GRADLE, M.D.
PETER KRONFELD, M.D.
MARK J. SCHOENBERG, M.D.
*ADOLPH POSNER, M.D.
*D. O. HARRINGTON, M.D.

W. P. WHERRY MEMORIAL

September 1, 1943

The expectations of the Committee regarding the financial response to the plea to establish a W. P. Wherry Memorial fund of $16,500.00 were somewhat dashed. Approximately $5,000.00 has been subscribed and it does not appear that more will be forthcoming.

The $5,000.00 already subscribed, invested in Government War Bonds, will bring in approximately $120.00 a year. It is recommended that the basic plan of the Committee for the Wherry Memorial Membership be modified to this extent:

1. The outstanding man of the year to be picked by the American Board of Otolaryngology.
2. At the annual meeting he be awarded a W. P. Wherry Memorial Membership consisting of paid-up initiation and paid-up annual dues for ten years.
3. This award to be made annually.

The Committee hereby recommends this course of procedure to the Council.

H. S. GRADLE, M.D., *Chairman*

* Inactive Members now in the Armed Forces.

REVISION OF THE CONSTITUTION AND BY-LAWS

(Contained in the Minutes of the Business Meeting for 1943 on pages 90 and 111.)

PAN-AMERICAN CONGRESS OF OTOLARYNGOLOGY

October 12, 1943

(Invitations were mailed to a number of otolaryngologists in Central and South America and Canada to attend a preliminary meeting of the Committee during the Academy convention in Chicago, October 10-13, 1943. The Secretary of the Academy also sent invitations to the same physicians to participate in the scientific program and to attend the annual banquet at which a special program on Inter-American Relations had been prepared. The full report of the Committee, containing a detailed history of the development of the Congress, will be published as a supplement to the TRANSACTIONS.)

W. L. BENEDICT, M.D.

JOINT COMMITTEES

AMERICAN COMMITTEE ON OPTICS AND VISUAL PHYSIOLOGY

September 1, 1943

This Committee has held three meetings in the last year. Of a number of matters considered, the following were acted on:

Arrangement has been made with the Council on Physical Therapy of the American Medical Association whereby the members of the Committee on Optics and Visual Physiology will act in an advisory capacity in all questions pertaining to apparatus used in the field of ophthalmology.

Action has been taken leading toward some recognition by the authorities of the Army and Navy regarding the abuses of followers of the Bates and similar systems which claim to enable rejected men to pass visual requirements of the Navy and Air Services.

The work to date on terminology in ocular motility and allied subjects has been written by Dr. Walter B. Lancaster and published in the American Journal of Ophthalmology, February, 1943. The terms and definitions as shown by the author are not final, but submitted for general consideration and discussion.

Due to the character and number of changes which have been made in military visual requirements since June, 1942, another paper entitled "Ophthalmic Requirements of the Military Services" has been written by Dr. Charles A. Bahn and submitted to the Archives of Ophthalmology for publication.

ALFRED COWAN, M.D.

AMERICAN ORTHOPTIC COUNCIL

August 20, 1943

Although greatly restricted in scope, the activities of the American Orthoptic Council have continued uninterrupted during the past year.

The last executive meeting of the Council was held in Chicago on October 11, 1942. At that time an examination was given to eight candidates. The total number of orthoptic technicians now holding the certificate of the American Orthoptic Council is 62. To this number will be added those successfully completing the examinations to be held in Chicago on October 9, 1943.

The American Association of Orthoptic Technicians has worked hard and faithfully to improve the status of orthoptics in this country. Among their activities are the collection and evaluation of all printed material relating to orthoptics, and the preparation of a brochure for parents of children with strabismus explaining in easy language the aims and methods of orthoptics. This brochure will be submitted to the American Orthoptic Council for editing before being published.

The proper military authorities have been informed of the present status and potentialities of orthoptics in relation to military needs.

Encouragement has been given to the establishment of training schools in areas where such are needed and where proper supervision can be exercised. We look forward hopefully to the efforts of Dr. Lawrence T. Post and his associates in St. Louis to develop a new school where excellent training may be received.

Personnel

No changes were made in the appointments to the American Orthoptic Council by the American Ophthalmological Society and the American Medical Association.

Officers

The officers for 1943 are LeGrand H. Hardy, president; Beulah Cushman, vice-president and Thomas D. Allen, secretary-treasurer.

Examinations

The dates of the 1943 examinations:

(1) First Part, written, September 9, 1943

(2) Second Part, oral-practical, at Children's Memorial Hospital, Chicago, October 9, 1943

A symposium on orthoptics will be conducted under the joint auspices of the American Orthoptic Council and the American Association of Orthoptic Technicians on October 10, 1943, during the Academy convention:

Recommendation

Your Committee respectfully recommends:

1. Continuation of the active sponsorship of the American Orthoptic Council by the American Academy of Ophthalmology and Otolaryngology and repetition of its annual grant of $200.
2. The re-appointment of Dr. John Hitz (now in active service) for a period of three years to succeed himself.

JOHN HITZ, M.D.
MURRAY MCCASLIN, M.D.
AVERY PRANGEN, M.D.

INDUSTRIAL OPHTHALMOLOGY

September 1, 1943

This Committee has been very active during the past year. The increased tempo of war-time production has brought to light the need of good vision and of proper protection from eye hazards for industrial workers. Ophthalmic service generally for industrial plants, and especially for the smaller ones, is inadequate in nearly all its branches — in protection, in testing, in correcting refractive defects, in treatment of eye injuries, in recording accident data, in placement of employees according to visual ability and skill, and in other related fields.

The activities of this Committee for the past year are indicated by the following summary:

1. The Committee has participated in educational programs in cooperation with the National Safety Congress, the Indiana Emergency Industrial Health Program, the Indus-

trial Council of the A.M.A., the Institute of the United States Public Health Nurses' Division of Industrial Hygiene and the Second Industrial Visual Institute at Purdue.

2. The various members of the Committee have worked with:

a. Committee on Inter-Professional Relations.
b. Eye-Saving Division of Conservation of Man Power.
c. Industrial Advisory Committee, National Society for the Prevention of Blindness.

3. The Committee has cooperated with the N. S. P. B. in the development of a correspondence course for industrial personnel, the production of a manual on industrial nursing and a goggle exhibit for the Academy meeting in October. It has cooperated with the National Safety Council of the A. M. A. in matters relating to vision, and with the Department of Labor, U. S. A.

4. There is in preparation a paper on the care and treatment of industrial eye injuries to be published and distributed by the A.M.A.

5. Associate members who have rendered valuable service have been added to the Committee.

6. The Committee has instituted a symposium on Industrial Ophthalmology to be given at a Sunday evening meeting during the convention of the American Academy of Ophthalmology and Otolaryngology.

7. It has issued instructions relating to epidemic keratoconjunctivitis and participated in a research project on this disease.

8. It has answered numerous inquiries — for example, on the use of safety glass and the field of usefulness of the Telebinocular.

9. Investigations are being continued on the following: Flash problems, a program of ophthalmic service for smaller plants, effects of radiation on vision, a simplified method of testing employees' vision.

10. The Committee has outlined the essentials of a basic industrial visual program. It has held consultations with war industries on specific visual problems.

11. The Secretary of the Committee has completed (now in press) a Treatise on Industrial Ophthalmology.

12. Some problems which the Committee has under consideration are:

a. The study of plans and proposals for rehabilitation of individuals with visual war disabilities both among the returning service men and among those injured in war industries. This entails study and work with the Veterans' Bureau, the study of State Security laws now being set up and of situations with insurance underwriters.
b. The study of ways and means whereby adequate ophthalmic service may be brought both to large and to small industries.
c. The preparation of a memorandum for the Industrial Health Council on examination of eyes as a part of the general physical examination already made by the Council.

It is recommended that the committee be continued.

ALBERT C. SNELL, M.D., *Chairman*
HEDWIG S. KUHN, M.D., *Secretary*

DR. BRAWLEY: The Council has approved for 1944 the continuance of the entrance fee of $20.00, dues for Junior Fellows $10.00, dues for Senior Fellows $5.00, dues for men in Military Service $5.00, and Life and Honorary Fellows to be exempt from paying dues.

Do you wish to act on this section?

PRESIDENT BABBITT: A motion is in order that this section on dues be accepted.

DR. POST: I move that the recommendations be accepted.

DR. C. H. MCCASKEY: I second the motion.

PRESIDENT BABBITT: It has been moved by Doctor Post, seconded by Doctor McCaskey, that these recommendations concerning dues be accepted.

The motion was put to a vote and carried.

DR. BRAWLEY: At the request of the American Board of Ophthalmology, Dr. Frederick C. Cordes, San Francisco, California, and Dr. Everett L. Goar, Houston, Texas, have been appointed by Council action to represent the Academy on this Board. Doctor Cordes shall serve for four years and Doctor Goar for one year to fill the unexpired term of Dr. W. L. Benedict who has resigned.

A motion is in order for this.

DR. RALPH A. FENTON: I move that the action be ratified.

DR. SHURLY: I second the motion.

PRESIDENT BABBITT: It has been moved by Doctor Fenton, seconded by Doctor Shurly, that this action be ratified.

The motion was put to a vote and carried.

DR. BRAWLEY: The Council recommends the nomination of Professor Olof Larsell, Ph.D., Sc.D., of the University of Oregon, to Honorary Fellowship in the Academy.

DR. SPENCER: I move that the recommendation be approved.

DR. FENTON: I second the motion.

The motion was put to a vote and carried.

DR. BRAWLEY: The Council recommends Dr. Ralph A. Fenton, Guest of Honor at the present session of the Academy, to Life Fellowship in the Academy.

We wanted to do Doctor Fenton all the honor in the world, but it happens that if we make him an Honorary Fellow, he loses his status and his vote, and he will be completely disfranchised.

DR. HARRIS P. MOSHER: I found that out after that was done to me and I didn't like it. It was put over on me. I never had that happen before.

PRESIDENT BABBITT: Is there a motion that the recommendation that Doctor Fenton be made a Life Fellow be approved?

DR. CARMODY: I move that Doctor Fenton be made a Life Fellow of the Academy.

The motion was seconded, put to a vote and carried.

DR. BRAWLEY: The Council re-appointed Dr. W. L. Benedict Editor-in-Chief of the TRANSACTIONS.

DR. FENTON: I move that the appointment be approved.

DR. SHURLY: I second the motion.

The motion was put to a vote and carried.

DR. BRAWLEY: It has been decided by the Council to hold the 1944 convention in Chicago the week beginning October 8. The final decision, however, is to be left to the discretion of the Program Committee at its January meeting because of possible unforeseen developments in the war situation.

PRESIDENT BABBITT: That does not need any action, but I think we should approve it.

DR. SPENCER: I move that the recommendation be approved.

DR. LLOYD: I second the motion.

The motion was put to a vote and carried.

PRESIDENT BABBITT: Next in order is the presentation of the list of candidates, which you all have. It has been carefully prepared and as far as we know is in order.

DR. SHURLY: I move that it be accepted and reported in the TRANSACTIONS.

PRESIDENT BABBITT: They must be voted into fellowship in the Academy. The list is accepted but do you make the motion that they be approved for fellowship in the Academy?

DR. SHURLY: I move that the candidates whose names appear on the list be approved for fellowship.

DR. FENTON: I second the motion.

The motion was put to a vote and carried and the following candidates were elected to fellowship:

Elected to Fellowship

Adix, H. Victor Jr., Lt. Comdr. (MC) U. S. Naval Hospital, Bremerton, Washington, ALR.

Allen, Wilmer L., Capt. (MC) Station Hospital, Camp Ellis, Ill., ALR.

Bair, Hugo L., Mayo Clinic, Rochester, Minn., Op.

Baldwin, Thomas M., 222 Brodhead Bldg., Beaver Falls, Pa., ALR.

Barrett, John A., 1304 Walker Ave., Houston, Texas, ALR.

Beil, Wallace C., 812 Maison Blanche Bldg., New Orleans 16, La., Op.

Berman, Harry L., 1142 Chapel, New Haven 11, Conn., ALR.

Berney, Irving Vincent, 31 Lincoln Park, Newark 2, N. J., ALR.

Bisno, Daniel, 4500 Olive St., St. Louis 8, Missouri, Op.

Blaydes, J. E., First Nat'l Bk. Bldg., Bluefield, W. Va., Op.

Bloch, Charles L., Lt. (MC) USNR Post Dispensary, Quantico, Va., Op.

Blumklotz, Bruno, 185 N. Wabash Ave., Chicago 1, Ill., ALR.

Bodenheimer, Ernst, 1212 Eutaw Pl., Baltimore 17, Md., Op.

Bower, Richard L., 1103 Grand Ave., Kansas City 6, Missouri, ALR.

Brady, Charles Henry, Sedalia, Mo., ALR.

Brooks, Clyde Montgomery, Capt. (MC) 105 S. Norman Ave., Carbondale, Ill., ALR.

Brooks, Jack E., 712 Grace St., Chicago 13, Ill., ALR.

Brown, Henry A., Mayo Clinic, Rochester, Minn., ALR.

Brown, Wilbur Max, Major (MC), 4th St., Mansfield, Ohio, Op.

Bryan, James H., 508 N. Grand Blvd., St. Louis 3, Missouri, Op.

Buchbinder, Murray W., Major (MC), 90th General Hospital, Fort Dix, N. J., ALR.

Budetti, Joseph A., Lt. (MC) 90 Sherman Ave., Mt. Vernon, N. Y., ALR.

Burston, Herschel, 1917 Wilshire Blvd., Los Angeles 5, Calif., ALR.

Byrnes, Victor A., Col (MC) School of Aviation Medicine, Randolph Field, Texas, Op.

Cammisa, James J. Vincent, Comdr. (MC) Navy 246, c/o Fleet, P. O., New York, N. Y., OALR.

Cannon, Harold J., 536 W. Wisconsin Ave., Milwaukee 3, Wis., ALR.

Cetner, John A., Lieut. (MC) 248 State St., Albany 6, N. Y., Op.

Chapman, E. J., 331 Haywood Bldg., Asheville, N. C., ALR.

Clapp, C. A., 513 N. Charles St., Baltimore 1, Md., Op.

Clark, Williard C., 530 Fidelity Bldg., Dayton 2, Ohio, Op.

Clothier, William L., 407-411 Kane Bldg., Pocatello, Idaho, Op.

Conley, John J., Capt. (MC) 313 Rowland Ave., Carnegie, Pa., ALR.

Constantine, Elizabeth Frederick, 927 Park Ave., New York 28, N. Y., Op.

Cooper, James H., 207 Toledo Med. Bldg., Toledo 2, Ohio, Op.

Cotten, Willis Frank, 202 State Street, McComb, Miss., ALR.

Cranston, William J., Jr. Capt. (MC), 175 Clinton Ave., Kingston, N. Y., Op.

Crawford, H. E., 139 Kinoole St., Hilo, Hawaii, T. H., Op.

Cunningham, Bernard Poland, Capt. (MC) 0486280, A.P.O. 464, 182nd Station Hosp., c/o Postmaster, New York, N. Y., ALR.

Cunningham, John C., Capt. (MC), 1200 Main St., Dubuque, Iowa, Op.

Cusick, Paul L., Major (MC), Mayo Clinic, Rochester, Minn., Op.

Damitz, John Cyril, Lt. (MC) USNR, 1036 Emma Ave., Akron 2, Ohio, OALR.

Daniel, Ruby K., Medical Arts Bldg., Dallas 1, Texas, Op.

Deems, Myers B., Major (MC) Fitzsimons General Hospital, Denver, Colorado ALR.

Dolowitz, David A., c/o A. Dolowitz, 70 W. 40th St., New York, N. Y., ALR.

Donlon, John J., 21 S. Village Ave., Rockville Center, N. Y., ALR.

Donovan, Richard Joseph, 1400 Hancock St., Quincy, Mass., OALR.

Downing, Arthur H.,Capt. (MC) Medical Induction Station, Fort Snelling, Minn., Op.

Drews, Leslie C., 516 Metropolitan Bldg., St. Louis 3, Missouri, Op.

Dubins, Henry B., 39-15 Main Street, Flushing, N. Y., Op.

Elliot, Alfred J., Night Vision Laboratory, Physics Bldg., McGill University, Montreal, Canada, Op.

Faggart, George Henry, 18 W. Oglethorpe Ave., Savannah, Georgia, ALR.

Faier, Herman I., Major (MC) Hoff General Hospital, Santa Barbara, Calif., Op.

Falls, Harold Francis, University Hospital, Ann Arbor, Mich., Op.

Fitzgerald, J. R., 30 N. Michigan Ave., Chicago 2, Ill., Op.

Follweiler, Frank L., 423 York Road, Jenkintown, Pa., ALR.

Foote, William D., Lt. (MC) Station Hospital, Hunter Field, Savannah, Ga., Op.

Forrester, Alex M., Capt. (MC), Roswell Army Air Field, Station Hospital, Roswell, New Mexico, ALR.

Fowler, Floyd J., Akron Clinic, 513 W. Market St., Akron 3, Ohio, ALR.

Fox, Samuel L., 1820 Eutaw Place, Baltimore 17, Md., ALR.

Frank, Herman W., Medical Bldg., Gadsden, Alabama, Op.

Frey, Walter D., 200 N. Upper St., Lexington 15, Ky., Op.

Gans, Jerome A., Lt. (MC), 3597 Bainbridge Rd., Cleveland Heights 18, Ohio, Op.

Gibson, William J., 650 Main St., New Rochelle, N. Y., ALR.

Gillis, Marion Howard, Jr., Salisbury, Md., Op.

Gissy, C. J., 717 Metropolitan Bldg., St. Louis 3, Missouri, Op.

Goldmacher, H. B., 113 Elmora Ave., Elizabeth 2, N. J., ALR.

Goodwin, Vernon L., 509 Central Tower, Youngstown, Ohio, ALR.

Griesemer, Z. Lawrence, 1145 E. Jersey St., Elizabeth 4, N.J., Op.

Guzek, Joseph T., 29 Greene Ave., Brooklyn 5, N. Y., Op.

Hagood, Daniel S., 822 First Nat'l Bk. Bldg., Montgomery 4, Ala., Op.

Haik, George M., Capt. (MC) 812 Maison Blanche, New Orleans 16, La., Op.

Haines, Henry L., Johns Hopkins Hospital, Baltimore, Md., ALR.

Hanser, Samuel Albert, Major (MC) 3109 Allen Ave., St. Louis 4, Missouri, Op.

Hauser, Maurice J., Major (MC) Station Hosp., Fort Custer, Mich., Op.

Hearn, Paul P., Capt. (MC) Station Hosp., B.A.A.F., Ft. Myers, Fla., ALR.

Hedemark, Norman G., Major (MC) 518 Eastman Bldg., Boise, Idaho, ALR.

Henry, L. L., 595 E. Colorado St., Pasadena 1, Calif., Op.

Hillyer, Ernest C., Comdr. (MC) 217 Bennett Street, Long Beach 3, Calif., Op.

Hoeltgen, Maurice M., 1607 West 51st St., Chicago 9, Ill., Op.

Hoffman, N. Fulmer, Jr., 2035 Delancey Pl., Philadelphia 3, Pa., ALR.

Holmen, Robert W., 1051 Lowry Medical Arts Bldg., St. Paul 2, Minn., ALR.

Holt, Helen, 720 North Michigan Ave., Chicago 11, Ill., Op.

Howard, H. Sargent, 418 Jefferson Bldg., Peoria 2, Ill., ALR.

Hoxie, Derrick, Lt. Cmdr., U. S. Naval Receiving Barracks, Shoemaker, Calif., ALR.

Hughes, William F. Jr., Johns Hopkins Hospital, Baltimore 5, Md., Op.

Hurwitz, Paul, Major (MC) Station Hosp., Camp Ellis, Ill., Op.
Isbell, Euclid A., 948 Forrest Ave., Gladsden, Ala., ALR.
Jarrott, James T., 131 Fulton Ave., Hempstead, N. Y., Op.
Jerome, Bourne, Major (MC) Station Hosp., Fort Sheridan, Ill., Op.
Jones, W. Yerby, 436 Sycamore St., Buffalo 4, N. Y., Op.
Jordan, Philip J., 910 Medico-Dental Bldg., San Jose 20, Calif., ALR.
Kennedy, Francis L., Missouri Theatre Bldg., St. Louis 3, Missouri, ALR.
Kershner, C. M., Watson Clinic, Brookings, S. D., Op.
Kimata, Harold T., Lt. (MC) Station Hosp. No. 2, Fort Bragg, N. C., ALR.
Kirch, Walter, 417 Bankers Trust Bldg., Des Moines 9, Iowa, ALR.
Klawans, Harold L., Capt. (MC) McCloskey General Hosp., Temple, Texas, ALR.
Koch, Harold J., 6235 S. Kedzie Ave., Chicago 29, Ill., Op.
Kos, Clair M., Major (MC) School of Aviation Med., Randolph Field, Texas, ALR.
Krieger, Alexander A., Major (MC) 2145 Wightman St., Pittsburgh 17, Pa. Op.
Lee, Wayne Robert, 613 Medical Arts Bldg., Burlington, Iowa, ALR.
Lempert, Julius, 119 East 74th St., New York 21, N. Y., ALR.
Leopold, Irving H., University Hospital, University of Pa., Philadelphia 4, Pa., Op.
Lewis, Samuel D., 49 Pearl St., Hartford 3, Conn., Op.
Lippmann, Otto, 406 Scarborough Bldg., Austin 22, Texas, Op.
Loder, L. L., 506 Hackley Union Nat'l Bk. Bldg., Muskegon, Mich., Op.
Longaker, Edwin P., 41 E. Montgomery Ave., Ardmore, Pa., ALR.
Loomis, George L., 310 Choate Bldg., Winona, Minn., ALR.
Loy, A. W., Comdr. (MC) Sch. of Aviation Med., U. S. Naval Air Station, Pensacola, Fla., Op.
McCall, J. W., 610 Rose Bldg., Cleveland 15, Ohio, ALR.
McGarry, H. Isabelle, 636 Church St., Evanston, Ill., Op.
McGowan, Joseph Francis, New Medical Bldg., Asheville, N. C., ALR.
McGuire, John, Peery Bldg., Bluefield, W. Va., ALR.
McKay, Edward D., Capt. (MC) 5th and Hazel, Texarkana, Arkansas-Texas, Op.
McLaughlin, R. C., 3780 Wilshire Blvd., Los Angeles 5, Calif., ALR.
McMurray, John S., Capt. (MC) 630 Washington Trust Bldg., Washington, Pa., ALR.
Macpherson, William A., Capt. (MC) 2701 East Florence Ave., Huntington Park, Calif., Op.
Mahoney, Frank A. Jr., Capt. (MC) 16 Harriet St., Springfield 7, Mass., ALR.
Maier, Marcus M., 513 Front St., Hempstead, N. Y., ALR.
Mann, Mortimer, 323 Hume-Mansur Bldg., Indianapolis 4, Ind., Op.
Marmelstein, Maurice, First Nat'l Bk. Bldg., Carbondale, Pa., Op.
Master, John R., Lt. (MC) Letterman General Hosp., San Francisco, Calif., ALR.
Matthews, John L., Major (MC) Sch. of Aviation Med., Randolph Field, Texas, Op.
Matzkin, Lloyd L., Capt. (MC) 117th Station Hospital, APO—929, San Francisco, Calif., ALR.
Maumenee, Alfred E., Johns Hopkins Hosp., Baltimore 5, Md., Op.
Maxwell, Hal W., Lt. (MC) Myra, Texas, Op.
Merz, Earl H., 55 E. Washington St., Chicago 2, Ill., Op.
Milder, Benjamin, 501 Humboldt Bldg., St. Louis 3, Missouri, Op.
Miller, A. Boyd, 1006 Professional Bldg., 429 Penn Ave., Pittsburgh, Pa., ALR.
Miller, Sidney N., 53 S. Hamilton St. Poughkeepsie, N. Y., ALR.
Minsky, Arthur, 2 E. 95th St., New York 28, N. Y., Op.
Mohun, Meade, 23 Second Ave., San Mateo, Calif., ALR.
Monaco, Louis, 233 S. San Fernando Blvd., Burbank, Calif., ALR.
Moore, Donald E., 1100 Euclid Ave., Syracuse 10, N. Y., Op.
Morrison, James D., Capt. (MC) 208 North 28th St., Billings, Mont., Op.
Morrone, Francis X., 117 S. 2nd Ave., Mt. Vernon, N. Y., ALR.
Moser, George P., Capt. (MC), 76 Willow St., Carlisle, Pa., ALR.
Newell, Frank W., Lt. (MC), 108 Gen. Hosp., New Orleans, La., Op.
Newton, Ray E., 1010 Reynolds Bldg., Jackson, Mich., Op.
Norris, Mary Alice, 404 Hume-Mansur Bldg., Indianapolis 4, Ind., Op.
Ogus, Alfred, School of Aviation Medicine, S.A.A.A.B., Santa Ana, Calif., ALR.
Overton, Jesse W., 324 East 41st St., New York 17, N. Y., ALR.
Owen, Walter E., 942 Jefferson Bldg., Peoria 2, Ill., ALR.
Patch, Orien B., Capt. (MC) U. S. A. A. F. T. T. C., Station Hosp., Lincoln Air Base, Lincoln 1, Neb., ALR.

Paton, Walter M., 919 Medical-Dental Bldg., Vancouver, B. C., Can., ALR.
Pattee, George L., 612 Republic Bldg., Denver 2, Colo., ALR.
Pearson, George J., 320 N .Third, Burlington, Iowa, ALR.
Penta, John J., 313 Washington, Reading, Pa., ALR.
Peter, Beverly K., Beckley Hospital, Beckley, W. Va., ALR.
Phillips, James R., Major (MC) Station Hosp., Camp Livingston, La., Op.
Pierce, George W., 490 Post St., San Francisco 2, Calif., PLS.
Platt, Edward S., Lt. Comdr. (MC) 4 Hamilton Ave., Auburn, N. Y., Op.
Podboy, August J., 211 S. George Street, York, Pa., ALR.
Portnoy, Samuel H., 19 Garfield Place, Cincinnati 2, Ohio, ALR.
Presnell, C. A., 634 N. Grand Ave., St. Louis 3, Missouri, ALR.
Pugmire, Ralph W., 2404 Washington Blvd., Ogden, Utah, Op.
Qualls, H. W., 1635 Exchange Bldg., Memphis 3, Tenn., ALR.
Quinlan, Joseph D., 44 Prichard St., Fitchburg, Mass., ALR.
Raines, Morris M., 34 N. Ash St., Ventura, Calif., ALR.
Ravin, Louis C., 316 Michigan St., Toledo 2, Ohio, Op.
Rech, Merrill J., Station Hospital, Randolph Field, Texas, Op.
Riter, Kersey Clyde, Capt. (MC) 69 E. First North St., Logan, Utah, ALR.
Robertson, E. Norris, Jr., 508 N. Grand, St. Louis 3, Missouri, Op.
Robison, J. M., 1304 Walker Ave., Houston, Texas, ALR.
Ronis, Bernard J., Capt. (MC) 7141 Elmwood Ave., Philadelphia 42, Pa., ALR.
Rosen, David M., 19 West 55th St., New York 19, N. Y., ALR.
Rosen, Emanuel, Capt. (MC) 692 High St., Newark 2, N. J., Op.
Rothenberger, Marvin K., 206 N. 8th St., Allentown, Pa., ALR.
Royer, Charles A., Lt. (MC) 431 College Ave., Alva, Okla., Op.
Rubin, Henry J., 611 N. Stanley Ave., Los Angeles, Calif., ALR.
Rubin, Herman G., Major (MC) 49th General Hospital, Chickasha, Okla., ALR.
Ryder, William H., 185 Church St., New Haven, Conn., Op.
Sacheroff, Louis, 422 Keith Bldg., Cleveland, Ohio, ALR.
Sadoff, Irvin E., Lt. (MC), 1045 N. Ave., Elizabeth, N. J., ALR.
Samponaro, Nicholas, 650 Main St., Hartford 3, Conn., ALR.
Sauer, John J. 30 East 40th St., New York 16, N. Y., Op.
Scheffler, Milton Morris, 58 E. Washington St., Chicago 2, Ill., Op.
Scholz, Roy O., Johns Hopkins Hospital, Baltimore 5, Md., Op.
Schwarz, Gerald T., 10515 Carnegie Ave., Cleveland 6, Ohio, Op.
Scobee, R. G., 2430 Bluebonnet, Houston 2, Texas, Op.
Scurti, Bernard V., 51 Harmon St., Brooklyn 21, N. Y., ALR.
Seip, Walter R., Capt. (MC) 429 Penn Ave., Pittsburgh 22, Pa. Op.
Shanedling, P. D., 310 S. Michigan Ave., Chicago 4, Ill., Op.
Simmons, J. Thomas, Lt. (MC) 316 W. 10th St., Erie, Pa., Op.
Sitney, Julian J., Lt. (MC) 58 E. Washington, Chicago, Ill., Op.
Smith, Leslie M., U. S. Marine Hosp., Baltimore 11, Md., Op.
Smith, Norman N., 291 Whitney Ave., New Haven 11, Conn., ALR.
Smith, Thomas T., Lt. (MC), Norfolk Naval Hospital, Portsmouth, Va., ALR.
Stauffer, Harry B., 234 Madison, Jefferson City, Missouri, ALR.
Stevenson, Walter D., Jr., W. C. U. Bldg., Quincy, Ill., ALR.
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Stoner, Donald D., 156 W. Pomfret St., Carlisle, Pa., ALR.
Strause, Harold L., 826 N. 5th St., Reading, Pa., ALR.
Strauss, Arthur, 130 Pavilion Ave., Long Branch, N. J., ALR.
Strickler, Alfred D., 26 N. 9th St., Lebanon, Pa., Op.
Stuard, Charles G., Major (MC) Medical Arts Bldg., Tulsa 3, Okla., Op.
Swan., Kenneth C., University Hosp., Iowa City, Iowa, Op.
Tangen, George M., University of Minnesota, Minneapolis 14, Minn., ALR.
Thacker, Edgar A., 602 W. University Ave., Urbana, Ill., ALR.
Thomas, Charles I., 10515 Carnegie Ave., Cleveland 6, Ohio, Op.
Thompson, William R., Major (MC) 281 S. Ashland Ave., Lexington 37, Ky., ALR.
Tinkess, Donald E., Major (MC) Ituri Towers, Greenwich, Conn., Op.
Tyler, Richard E., The Norwood Clinic, 2501 N. 16th Ave., Birmingham 4, Ala., ALR.
Van Wien, Stefan, Lt. (MC) 104 S. Michigan Blvd., Chicago, Ill., Op.

Weaver, Emerson M. F., Major (MC) 18 -Repl. Wing, Salt Lake City, Utah, Op.
Webb, M. A., 4500 Olive St., St. Louis, Mo., ALR.
Wellford, B. R., 510 Medical Arts Bldg., Richmond 19, Va., ALR.
Welt, Bernard, 135 New York Ave., Brooklyn, N. Y., ALR.
Wesson, Thomas W., 124 S. E. First, Evansville 8, Ind., ALR.
Wiest, Philip R., Capt. (MC) 1036 Franklin St., Reading, Pa., ALR.
Wilcox, Malcolm B., 5 West 21st St., Kearney, Neb., ALR.
Williams, John W., Lt. (MC) Medical Arts Bldg., Springfield, Missouri, ALR.
Williams, Russell I., 422 Hynds Bldg., Cheyenne, Wyo., ALR.
Wilmes, Malcolm N., Major (MC) 82nd General Hospital, Baxter General Hospital, Spokane, Wash., ALR.
Winer, Albert S., 1835 Eye St., N. W., Washington, D. C., ALR.
Wojniak, Frank, 4649 S. Ashland, Chicago 9, Ill., ALR.
Wolf, Lewis R., 255 S. 17th St., Philadelphia 3, Pa., Op.
Woolfolk, William L., 115 E. 4th St., Owensboro, Ky., ALR.
Work, Walter P., Capt. (MC) 384 Post St., San Francisco, Calif., ALR.
Zeman, Michael S., Capt (MC) Station Hospital, Camp Phillips, Kansas, ALR.

PRESIDENT BABBITT: The second report from the Senior Member of the Council will now be presented.

DR. BRAWLEY: The Council has approved the action of the Executive, Research and Finance Committee in nominating the following Fellows as officers for the ensuing year:

President.....Lawrence T. Post, M.D.
St. Louis, Mo.

President-Elect.....Gordon B. New, M.D.
Rochester, Minn.

First Vice President..C. Stewart Nash, M.D.
Rochester, N. Y.

2nd Vice Pres.....Harold F. Whalman, M.D.
Los Angeles, Calif.

3rd Vice Pres.....Chevalier L. Jackson, M.D.
Philadelphia, Pa.

Executive Secretary-
Treasurer.....William L. Benedict, M.D.
Rochester, Minn.

Secretary for
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New York, N. Y.

Secretary for
Otolaryngology....O. E. Van Alyea, M.D.
Chicago, Illinois

Secretary for Instruction—
Ophthalmology....A. D. Ruedemann, M.D.
Cleveland, Ohio

Secretary for Instruction—
Otolaryngology and Maxillofacial
Surgery.....Dean M. Lierle, M.D.
Iowa City, Iowa

Secretary for Home
Study Courses.....Harry S. Gradle, M.D.
Chicago, Illinois

Secretary for
Public Relations..Erling W. Hansen, M.D.
Minneapolis, Minnesota

Councillor.....J. Milton Robb, M.D.
Detroit, Michigan

It was regularly moved, seconded and carried that the slate be approved and that the officers be elected.

PRESIDENT BABBITT: I should like to ask Doctor Shurly and Doctor Spencer if they will escort the new President forward, to be recognized by the Chair.

Dr. Lawrence T. Post was escorted to the rostrum by Doctors Shurly and Spencer.

PRESIDENT BABBITT: Doctor, we are proud to recognize you as the new President. As this report was being read I had a fleeting vision that all my happy times as President were past.

PRESIDENT-ELECT POST: Mr. President and Fellows of the Academy: The brevity of my remarks are no indication of the depth of my appreciation. I cannot begin to say how deeply I do feel this honor. I can only promise to help the Academy in the coming year to the best of my ability.

PRESIDENT BABBITT: Will Doctor Coates and Doctor Carmody escort Doctor New, the President-Elect for 1944, to the rostrum.

Dr. Gordon B. New was escorted to the rostum.

PRESIDENT BABBITT: Doctor New, it is with great delight that I recognize the action of the Council and the approval of this body in electing you to this most important office this year.

DR. NEW: Thank you, Doctor Babbitt. I appreciate the honor very much of being associated with the men who have done so much for the Academy over these years. I think it is recognized that the Academy is the greatest teaching society we have today, and it is going to be a great pleasure for me to try to

do my part to keep pace with those who have gone before.

PRESIDENT BABBITT: I should like to ask the Third Vice President, Dr. Chevalier L. Jackson, and the new Councillor, Dr. James M. Robb, if they will arise.

The President's duties, I am told, do not conclude until the first of January, so I shall not put the gavel into the hands of the new President whom we have elected today.

I want, in closing, to express my appreciation for the splendid support of the members of the Executive Committee and the other members of the Academy. I want particularly to thank the Secretary-Treasurer who has taught me much during my association with him. I want to thank Doctor Coates, Doctor Spencer and Doctor Myers who have been so loyal in their special help to me at these meeting times. It has been a great pleasure. As our Secretary-Treasurer said, "In spite of all the busy time, I have loved it." Thank you.

Our meeting is adjourned.

The meeting adjourned at six-thirty p.m

The Committee on Revision of the Constitution and By-Laws proposes changes in the Constitution as follows:

Article I, which reads:

The name of this society shall be THE AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

to read:

The name of this society shall be AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Article III, which reads:

FELLOWSHIP

The membership of this Academy shall consist of Active Fellows and Honorary Fellows, and their qualifications shall be as follows:

(a) A candidate for Active Fellowship shall be a member in good standing in his or her local, county, or state society, and shall have been practicing medicine in the special branches of eye, ear, nose or throat, exclusively, for at least one year. The candidate shall present the regular application blank of the Academy duly filled out and endorsed by two Active Fellows of the Academy, at least one of whom shall be a resident of his own city or state (except when there are no members of this society in the state), and accompanied by the entrance fee of ten dollars ($10.00). Such application shall be filed with the Secretary at least six weeks before the annual meeting. A list of the applicants shall be sent to the Fellows with the final notice of the annual meeting.

An applicant for membership must be approved by the American Board for Ophthalmic Examinations, or by the American Board of Examiners in Otolaryngology. When, however, an applicant for Fellowship fails to pass the examination of the American Board for Ophthalmic Examinations or of the American Board for Otolaryngology, he shall not be eligible for election until he has passed the Board before which he failed. If an applicant shall fail to qualify for Fellowship within three years after his application has been filed with the Secretary, the application fee is forfeited—however, the applicant may renew his application for an additional three years without paying an additional fee. Application fee is not returnable unless through action of the Council after the candidate is refused election.

Application shall be presented to the Council and, after its approval, shall be submitted to a general vote of the Academy at any annual meeting. A three-fourths affirmative vote shall constitute an election.

When a member is elected to Fellowship in the Academy, with the certificate of one of the national examining boards, he shall be classified in the official membership list of the Academy as practicing the specialty for which he holds the certificate, and shall not be classified as both an ophthalmologist and otolaryngologist until he shall have received the certificate of both Boards.

(b) Any Doctor of Medicine who has materially aided the progress of medicine in its relation to diseases of the eye, ear, nose or throat, may, on nomination by two Fellows and recommendation of the Council, and by a unanimous vote of the Fellows present at an annual meeting, be elected an Active Fellow.

(c) Anyone who has signally aided the progress of medicine in its relation to diseases of the eye, ear, nose or throat, may, on

recommendation of the Council and by a unanimous vote of the Academy at any annual meeting, be elected an Honorary member.

(d) The Council, for sufficient reasons, may recommend members to Life membership without dues, and a unanimous vote of the members present shall constitute an election.

(e) Expulsion. Membership in this Association shall continue only so long as the member shall by act or conduct reflect no discredit upon this Association, and an immoral or unethical act or conduct, including any violation of the Code of Ethics of the American Medical Association, shall be deemed grounds for expulsion. No member may be expelled, however, until after written charges have been prepared by the Judicial Committee, based upon either direct knowledge of the Committee or information brought to it indirectly, and such charges shall be presented to the Executive Committee, and such Committee shall give the member notice in writing, by registered mail, not less than thirty days beforehand, of the time and place of a hearing on said charges. The decision of a majority of said Executive Committee after such hearing, whether the member charged be present or represented or otherwise, shall be final, unless such member, if found guilty and expelled, shall appeal to the Academy. In such event he shall be entitled to such hearing at the next annual meeting of the Academy, and a majority of the Active Fellows present at such meeting shall determine whether the action of the Executive Committee shall be sustained or the member be re-instated. Such hearing shall, at the option of the Academy, be conducted in a secret session.

No member expelled shall be eligible to re-election until after three years from the date of expulsion.

to read:

FELLOWSHIP

The Academy shall consist of all members of the Society, such members to be designated as Active Fellows and Honorary Fellows.

(a) *Active Fellowship*

A candidate for Active Fellowship shall be a member in good standing of his or her local, county, state and/or national medical society and must be certificated and approved by the American Board of Ophthalmology, the American Board of Otolaryngology or the American Board of Plastic Surgery. When a candidate is elected to Fellowship in the Academy, he shall be classified in the Directory of the Academy as practicing the specialty or specialties for which he holds a Board certificate.

The application blank provided by the Academy shall be duly filled out by the candidate and filed, together with the current application fee, with the Executive Secretary-Treasurer. The application shall carry the endorsement of two Fellows of the Academy, at least one of whom shall be a resident of the candidate's home state or province (except where there are not members of this Society resident in the state or province).

If an applicant shall fail to qualify for Fellowship within three years after his application has been filed with the Executive Secretary-Treasurer, the application fee is forfeited — however, the applicant may within the next year renew his application for an additional three year period without paying an additional fee. Application fee is not returnable except by order of the Council after the candidate is refused election.

At least four weeks before the annual meeting a list of all applications filed with the Executive Secretary-Treasurer shall be sent to the Fellows with the official call of such meeting. Applications so listed shall be presented to the Council and after its approval, shall be submitted to the Active Fellows of the Academy for voting at the next annual meeting. Other applications not included in said list shall not be submitted by the Council for vote of the Active Members of the Academy until after on annual meeting. A three-fourths affirmative vote of all Active Fellows present and voting shall constitute an election.

Any doctor of medicine, who has materially aided the progress of medicine in its relation to diseases of the eye, ear, nose or throat, may, on nomination by two Fellows and recommendation of the Council, and by a unanimous vote of the Fellows present at an annual meeting, be elected an Active Fellow.

(b) *Honorary Fellowship*

Anyone who has signally aided the progress of medicine in its relation to diseases of the eye, ear, nose or throat, may on recommendation of the Council and by a unanimous vote of the Academy at any annual meeting, be elected an Honorary Fellow. Honorary Fellows shall not be required to pay any fees or dues and shall enjoy all the privileges of Active Fellows except those of voting and holding office.

(c) *Expulsion*

Fellowship in this association shall continue only so long as the Fellow shall by act or conduct reflect no discredit upon this Society. Any immoral or unethical act or conduct, including any violation of the Principles of Ethics of the American Medical Association, shall be deemed grounds for expulsion.

Charges leading to expulsion of a Fellow shall be submitted to the Judicial Committee for investigation and consideration. If the Judicial Committee by unanimous vote declares that the evidence in support of the charges is not sufficient to warrant expulsion that Committee will so report to the Council and the decision of the Committee shall be final and the charges dropped. If, however, the Judicial Committee rules that the evidence in support of the charges is sufficient to warrant expulsion, the charges shall be made in writing by the Judicial Committee and submitted, together with a transcript of all evidence, direct and indirect, to the Council. The Executive Secretary-Treasurer shall give the Fellow notice in writing, by registered mail, not less than thirty days beforehand, of the time and place of a hearing on such charges, and the Fellow so charged shall be given opportunity to be heard and present his defense thereto. The decision of the majority of the Council after such hearing, whether the Fellow charged be present or represented or otherwise, shall be final. A Fellow expelled shall not be eligible to re-election until after three years from the date of expulsion.

Article IV shall be as follows:

PROPERTY

The Academy shall have authority to purchase, accept, or otherwise acquire, hold, own, mortgage, pledge, sell, exchange, assign, transfer, or otherwise dispose of, invest and deal in real and personal property of every class and description.

Article IV, which reads:

OFFICERS

The officers of the Academy shall be:
President
President-Elect
First Vice President
Second Vice President
Third Vice President
Comptroller
Executive Secretary-Treasurer
Secretary for Ophthalmology
Secretary for Otolaryngology
Secretary for Instruction
Associate Secretary for Instruction
Editor of TRANSACTIONS
Board of Councillors

These officers shall be elected annually, except the Councillors, who are to serve for four years, one to be elected each year.

The Council shall consist of the President, who shall be the Chairman, President-Elect, First Vice President, the three most recent past Presidents, and the four active fellows who shall have been elected to serve four years. The Comptroller and secretarial group shall be ex-officio members of the Council.

Nominations for officers are presented to the Academy by the Council. Other nominations may be made by a written petition signed by five (5) Active Fellows.

The nominations shall be made and the election held at an annual meeting. A majority vote cast by the Active Fellows present at such a meeting shall constitute an election.

shall become Article V, to read:

OFFICERS

The officers of the Academy shall be:
President
President-Elect
First Vice President
Second Vice President
Third Vice President
Executive Secretary-Treasurer
Secretary for Ophthalmology
Secretary for Otolaryngology
Secretary for Instruction in Ophthalmology
Secretary for Instruction in Otolaryngology and Faciomaxillary Surgery
Secretary for Home Study Courses
Secretary for Public Relations
Editor-in-Chief of TRANSACTIONS
Councillors, of whom there shall be four

These officers, except the Councillors, shall be elected yearly. The Councillors are to serve for four years, one to be elected each year. The nomination and election of officers shall take place at the annual meeting. Only Active Fellows in good standing are eligible to hold office and to vote on matters presented at the annual meeting. Nomination of Fellows for officers shall be made by the Council and the election shall be held at the annual meeting of the Academy. A majority vote cast by the Active Fellows present at such a meeting shall constitute an election. Other nominations may be made by written petition signed by five (5) Active Fellows.

(Old) Article V shall become Article VII. (Amendments), otherwise unchanged.

(New) Article VI shall be:

THE COUNCIL

Section 1.

The Council shall be the governing body of the Academy, and shall consist of the President, who shall be the Chairman, the President-Elect, the Vice Presidents, the three most recent Past Presidents, the senior of whom shall be known as the Senior Member of the Council, the Secretaries of the Academy, the four Active Fellows who have been elected Councillors and the President of the Honor Section.

Section 2.

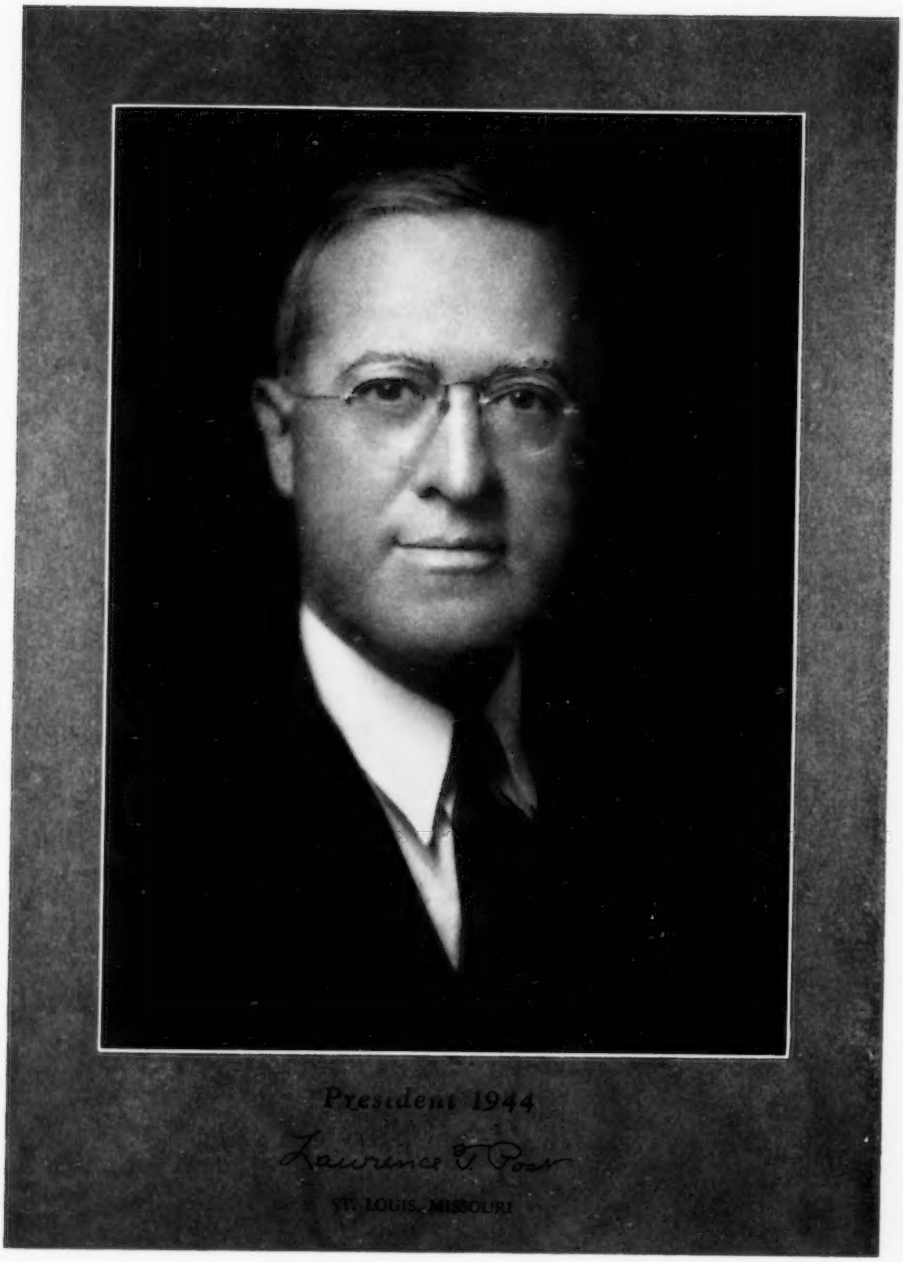
The Council shall have and control the management of the affairs and funds of the Academy, fix entrance fees and dues of Fellows, fix the date and place of the annual meeting, create and authorize the appointment of standing committees to conduct the affairs of the Council, have supervision of all activities and business affairs of the Academy and transact all business not otherwise provided for that may pertain to the Academy. Ten members of the Council shall constitute a quorum for the transaction of business.

IMPORTANT ANNOUNCEMENT

Effective January 1, 1944, the address of the Executive Office of THE AMERICAN BOARD OF OPHTHALMOLOGY will be: P. O. Box 1940, Portland 2, Maine. Please address all Board correspondence to this new address.

1944 Examinations

New York City, June 3 and 4
Chicago, October 5, 6 and 7



President 1944

Lawrence E. Poor

ST. LOUIS, MISSOURI

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(Term of Office Began January 1, 1944)

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St. Louis, Missouri

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American Academy of Ophthalmology and Otolaryngology

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ETIOLOGY OF SO-CALLED CHRONIC SIMPLE GLAUCOMA
Possible Role of Focal Infection, Especially of the Nasal Accessory Sinuses

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ALTHOUGH recent studies and contributions to the literature have added much to the knowledge of so-called chronic simple glaucoma, the fact remains that the etiology of the disease is still unknown. Numerous theories have been advanced but none is adequate, either from the experimental or clinical viewpoint. Study of pathologic material has met with scant success, for the enucleated eye usually shows only the result of an atrophic degenerative process and gives few clues to etiology. Lack of definite knowledge in regard to etiology forces ophthalmologists to resort to treatment which is symptomatic and palliative.

In the daily observation of patients with glaucoma, and in the study of literature dealing with this disease, one is constantly reminded of Lagrange's statement that "the eye attacked by hypertension is a sick eye in a sick body." Because of this, one finds the attention of the ophthalmologist becoming less confined to subjective symptoms and treatment, and more concerned with the etiology of the disease. In regard to primary glaucoma, investigation is being directed toward a search for the underlying causes, whether local or systemic. In this we are in agreement with the general trend of thought, which is to consider so-called "primary" glaucoma as possibly secondary in nature. Focal infection has sometimes been suggested as a probable etiologic factor. In this connection our interest has been drawn particularly to the possible role of sinus infection and nasal pathology.

This study was undertaken because of the apparent relationship between sinusitis and glaucoma, especially in cases of unilateral involvement, which we observed from time to time. We realize that the possibility of such a relationship may not be readily accepted and, therefore, expect and encourage criticism, especially concerning conclusions which seem to be supported by insufficient data. By correlating clinical and experimental data obtained from our own records and from the literature, it is hoped that this paper may arouse interest and stimulate study of chronic infection, particularly of the nasal accessory sinuses, as an etiologic factor in patients with chronic simple glaucoma. Before presenting our clinical studies, a brief discussion of focal infection in general and sinus infection in particular seems desirable.

FOCAL INFECTION IN RELATION TO EYE DISEASES WITH SPECIAL REFERENCES TO SINUS INFECTION

Among the foci mentioned frequently in the literature as being associated with eye diseases are the teeth and gums, tonsils, nasal accessory sinuses, the gallbladder, the intestinal tract and the gen-

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ito-urinary tract. Of these, the first three have probably received the most attention, particularly the teeth and tonsils. Our own experience bears out the conclusions of MacKenty,[25] Rutherford[32] and others, who found that sinusitis is often associated with infected tonsils or teeth which directly "feed" the sinus infection and thereby produce a secondary focus which may be more toxic than the primary one.

Some of the ocular conditions, in which sinusitis has been mentioned as a possible etiologic agent are uveal disease, optic neuritis, retrobulbar neuritis and orbital infection. Although some investigators believe that too much attention may be focused on the sinuses, thereby causing other serious conditions to be overlooked, it would appear from the literature that the tendency has been to ignore the condition of the sinuses or to give them only minor consideration. This has also been true of other focal infections, the accidental or incidental removal of which disclosed their probable relationship to the ocular disturbance.

The etiologic role of focal infection in chronic simple glaucoma has been suggested by a number of authors. According to Theobald,[35] edema and evidence of past attacks of edema are found histologically in eyes with increased tension, and, therefore, one may find an explanation of primary glaucoma in conditions that produce edema. Acharya[1] considers the disease to be caused by auto-intoxication and has found the average basal metabolic rate, blood urea, pulse rate and erythrocyte sedimentation rate to be higher than normal in glaucomatous patients. By analyzing the pathologic changes in the circulatory, nervous and endocrine systems, Massoud[27] demonstrated the presence of toxins or of a toxin in cases of glaucoma, and suggested that prevention and treatment of primary glaucoma be carried out on the basis of biochemistry and bacteriology. Risley[29] believed that the cause of glaucoma might be found in underlying systemic infections and should be regarded as an ocular manifestation of either a general or endogenous toxemia on the one hand, or a local or exogenous infection on the other hand. According to Allen,[2] focal infection, even though it may have no direct bearing on the glaucomatous process itself, lowers the general resistance of the body and, therefore, should be eliminated. Sugar[34] mentions hyperemia of the ciliary body due to upper respiratory infections as one of the possible causes of angle blockage which might bring on a glaucomatous attack.

Oral sepsis has been suggested as a possible cause of glaucoma. Elliot[17] cited a case in which the patient's visual fields and vision diminished rapidly in spite of normal tension and free filtration after successful trephining. Search for a possible focus revealed suspicious teeth which were removed, after which there was an early improvement in visual fields and vision. Dowling[16] believes that in certain cases of glaucoma the cause may be found in a toxic principal originating in the gallbladder. Allen[2] has found that constipation complicates the picture in glaucoma far more frequently than one would suppose from reading the literature. This suggests the possibility of intestinal infection, a condition noted in a high percentage of our own cases. A logical reason for this may be found in certain bacteriologic data[12,23,24] which show a decided correlation between upper respiratory and intestinal infections, the latter in all probability being the result of constant ingestion of bacteria or their toxins. Bacteriologic studies of cases of so-called intestinal intoxication have frequently revealed the presence of pathogenic streptococci in the feces, associated with a marked reduction from normal in the number of *B. coli*. This in-

dication of an inhibiting effect of the streptococci on the growth of *B. coli* is further suggested by the fact that, whereas rectal implants of *B. coli* suspensions do not result in a permanent increase in the number of colon bacilli, the administration of autogenous streptococcal vaccines has, in some cases, been effective in reducing or eliminating the fecal streptococci. Simultaneously, there has been a noticeable rise in the number of *B. coli*. Almost invariably, these cases of streptococcal intestinal infection are associated with a marked nasal or oral infection, as evidenced by clinical signs and symptoms and the presence of great numbers of pathogenic organisms.

As far back as 1830, Fabini[19] associated chronic simple glaucoma with arthritis, and our observations tend to support this viewpoint. Evans[18] has mentioned the nose, sinuses, tonsils and teeth as underlying factors in vascular disturbance of the eye. It might be well to state at this point that we agree that any or all of these foci may be associated with chronic simple glaucoma and possibly may be etiologic factors. Frequently more than one focus may be present. It is also probable that glaucoma is not the only disturbance caused by these foci, which would account for the number of cases in which there seems to be a general breakdown in physiologic functions, including disturbance of ocular function.

While it is true that sinus disease is almost universal, it is only occasionally that it appears to affect the eye, thus suggesting predisposing or contributing factors. MacKenty[25] believes that sinusitis varies "tremendously" in its toxicity, due more to the local and general resistance of the individual than to the nature of the infection. While a lowered resistance may be partially attributed to heredity, there is no doubt that such factors as extreme fatigue, toxemia and worry or shock play an important role. Under these conditions it is no wonder that the surgery of sinuses is so frequently discouraging; in some cases the patients have undoubtedly become sensitized to their own bacteria and we agree with MacKenty[25] that it is often necessary to resort to immunotherapy.

There seems to be a consensus of opinion that in the intra-ocular complications associated with sinus disease it is frequently the posterior ethmoid and sphenoid sinuses which are involved, and our experience is in accord with this current belief. Also, it is not usually the chronic purulent type which causes trouble, but rather the hyperplastic or occult type. The latter is potentially a greater source of danger because it is more likely to cause local or systemic invasion than the purulent type, which is to a great extent walled off from the blood and lymph circulation. Further, the occult type often gives very little evidence of its existence either by direct examination or by roentgenographic examination. These ideas have proved to be valid in many of our patients, so far as the relation of any focus of infection can be proven to be a factor in any organ not directly in contact with a focus. Therefore, we make unusually careful search for chronic hyperplastic disease in the sinuses and rely more on history and bacteriology in making an early tentative diagnosis than on examination, roentgenography and even laminography. Later events have frequently confirmed our tentative diagnoses.

SINUS DISEASE IN RELATION TO GLAUCOMA

So far as we have been able to ascertain, there have been only casual reference in the literature[18,29,34] concerning sinus disease as a possible etiologic factor in chronic simple glaucoma, and apparently little has been done to determine whether or not there is a definite association between the two diseases.

The difficulties of proving a definite relationship between the simultaneous occurrence of sinus disease and chronic simple glaucoma from the experimental or even the clinical standpoint seem to be unsurmountable.

To begin with, diagnosis of the ocular condition often presents a problem. Judging from a comparison of the clinical diagnosis and the pathological diagnosis in 437 cases of uveitis,* it would seem as though the clinical diagnosis of simple glaucoma does not always exclude secondary glaucoma. In 38 of these 437 cases the clinical diagnosis was primary glaucoma, whereas the pathological diagnosis was uveitis of undetermined etiology, with secondary glaucoma. Since clinical diagnoses do not appear to be exact in distinguishing between these diseases, and because uveitis is usually caused by pathogenic organisms, we naturally suspect the latter of having an effect on the eye in this group of cases.

The clincial picture also is greatly complicated by the high incidence of sinus disease in which no ocular involvement is demonstrable, or of bilateral sinusitis in which the ocular disease is unilateral. For this reason we have been particularly interested in those cases in which both the sinus and eye were unilaterally involved on the same side, or in which the involvement on one side overshadowed the other in severity. We reasoned that if a study of chronic simple glaucoma and sinus disease indicated a high incidence of more advanced glaucoma on the side of the more active or more advanced chronic sinus infection, that the probable relationship of the two conditions would, to say the least, be highly suggestive. Equally suggestive are the results frequently observed when the sinuses of glaucoma patients are successfully treated. For example, writing in reference to one of our glaucoma patients, Dr. L. T. Perrault said that he had noted "a reduction in tension followed tamponing of the nose with argyrol packs, reducing congestion and cleaning the whole upper vault of the nose (especially of the ethmoidal area), and, as they reach back to the sphenoidal opening, affecting it in the same way. As I have seen them markedly relieve choked disk due to sphenoidal infection, I think they would also act favorably upon an increased tension due to some infection in that area, which in this case was not evident upon examination."

That sinus infection may increase intra-ocular tension without producing any other symptom of glaucoma was suggested by one of our cases, not included in this study, in which ocular hypertension, though present, fluctuated above normal, finally became stationary at normal levels and chronic glaucoma never developed. Although tension rose as high as 35 mm. Hg. (Schiötz) in the left eye and 32 mm. Hg. (Schiötz) in the right, the field findings were more typical of sphenoid sinusitis than of glaucoma. During observation over a period of years the sinuses improved and there was a coincidental improvement of the tension and fields. However, although sinusitis may occasionally produce some one of the symptoms of glaucoma, its relationship to the disease complex known as chronic simple glaucoma is of greater importance.

In studying chronic, ocular inflammatory diseases, which over a long time affect only one eye, we have learned to search most carefully and repeatedly for a focus of infection on the same side as the eye lesion.

The association of uveitis and secondary cataract with unilateral chronic sinus infection has frequently been observed in our practice, and appears to be one of the most striking facts suggesting focal infection as the cause of chron-

* Data collected through the courtesy of Colonel J. E. Ash and Mrs. Helenor Wilder.

ic uveitis. Judging from our experimental work and that of others,[4,5,6,9,31] focal infection as a cause of eye lesions will not be proven definitely by experiments alone, unless the work can be carried out on man. Therefore, statistical clinical investigation is highly important.

CLINICAL STUDY

In Table I are summarized our findings in 53 cases of chronic simple glaucoma from private practice, on which we had sufficient data for the purposes of this investigation.

Prior to this study, there was no anticipation of the proportion in which there might be an associated nasal or sinus condition of etiologic significance, and there has been no attempt to select cases that might support a preconceived theory. However, until two years ago, we had not planned to correlate our data and, therefore, many of our records could not be included because they did not contain the information necessary for a survey of this kind. Other records had to be eliminated because of certain factors that might make the diagnosis of chronic simple glaucoma questionable. Five cases were omitted because dental, tonsillar or intestinal infection seemed to be so obviously associated with the glaucomatous condition that other factors appeared to be secondary or incidental.

Diagnosis was made on the presence of one or more of the generally accepted symptoms of chronic simple glaucoma. Most of the patients included in this report have been under our observation for at least three years, some for as long as 22 years.

Of the 53 cases included in this study, in 19 the glaucomatous condition was more pronounced in the left eye, and signs or symptoms of nasal involvement were either confined to the left side or more exaggerated on that side. Of these 19 cases there was one in which the presence of highly pathogenic bacteria in the left nasal membranes was the only indication of probable nasal pathology. In another, in the right eye there previously had been noted marked glaucomatous activity in association with involvement of the right ethmoidal and sphenoidal sinuses, but when observed by us, activity in the left eye was associated with involvement of the left frontal and maxillary sinuses.

In 18 cases the glaucomatous condition in the right eye was associated with nasal pathology on the right side, although in one case the sinus condition may have been secondary to a severely infected right tonsil.

In 10 cases one eye was more involved than the other, but nasal or sinus pathology was either bilateral or slightly greater on the side of the less affected eye.

In six cases both eyes were about equally affected by glaucoma and nasal pathology was about the same on both sides.

Reviewing the data, we find that although the evidence of a possible relationship between unilateral ocular activity and nasal pathology is not in all cases as convincing as might be desired, the fact that 37 of 53 cases (70 per cent) showed greater nasal and ocular involvement on the same side is highly suggestive. The findings are less convincing in the remaining 16 cases (30 per cent) in which nasal and ocular involvement was either bilateral, or in which the one eye was more affected than the other, in spite of bilateral nasal pathology. To offset this, however, is the fact that in no instance were marked signs and symptoms of glaucomatous activity in one eye associated with marked involvement of the nasal accessory sinuses on the opposite side.

In the 34 cases in which the bacteriology of the nasal tract was studied, the findings were not particularly signifi-

TABLE I.

Survey of 53 Unselected Cases of Chronic Simple Glaucoma in Relation to Nasal Pathology.*†

PATIENT AGE	SITE OF GLAUCOMA	EYE MOST AFFECTED	SINUSES			TENSION	FIELDS	MAXIMUM VISUAL ACUITY	NEAR POINT OF ACCOMMODATION	REMARKS
			EXAMINATION	BACTERIOLOGY	ROENTGEN RAY					
1 RD 39	O.D. O.S. (later)	O.D.	Post-nasal drip. Deflected septum right. Later pain and blurriness O.S. associated with left sinus exacerbations.	Pathogenic Strep. & Staph. in nasal culture.		Markedly increased O.D.	Consistently contracted O. D. O. S. normal until recent left sinusitis.	Worse O. D., especially during sinus attacks.	Markedly subnormal O. D. Improved to 240/3M later but always markedly more remote than O. S.	Deep cupping O. D. No cupping O. S.
2 MD 55	O.U.	O.S.	Left middle turbinate against septum, dry and red. Right turbinate normal.	Coliform bacteria in both nostrils.	Bilateral posterior ethmoiditis. Antra involved, left more than right.	O.S. higher than O.D.	Left definitely more constricted than right.	Left markedly poorer than right.	Left more remote than right.	Deep cup in nerve O.S. No cup in nerve O.D.
3 AC 64	O.U.	O.D.	Septum deflected. right more than left. Nasal blockage in AM, right more than left.	Pathogenic S. albus in both nostrils.		O.D. frequently than O.S.	O.D. worse than O.S. Improved with nasal improvement.	Same O.U.	O.S. more remote than O.D. O.S. glaucoma worse before observation here.	
4 JPG 70	O.U.	O.S. O.D. (later)	Septum deviated to left.	Pathogenic Strep. left nostril, no pathogenic bacteria recovered from right nostril.	Left frontal sinuses small and cloudy. Left antrum slightly cloudy. Left sphenoid slightly cloudy. Right sinuses clear.	O.S. consistently much higher than O.D. until flare-up in O.D. later.	O.S.—Small field to light, definitely worse than O.D.	O.S. definitely poorer than O.D.	O.S. consistently more remote than O.D.	No vitreous opacities when first observed, but numerous and large in O.S. later. Larger in O.D. when flare-up occurred in that eye.
5 WPC 33	O.U.	O.D.	Chronic nasal obstruction.			Always higher O.D. before trephine.	O.D. definitely worse than O.S. O.S. almost normal.	O.D. markedly decreased. O.S normal.	O.D. much more remote than O.S.	Marked cupping O.U. but O.D. more than O.S.
6 MC (Age not given)	O.U. (Incipient)	O.S. (slightly more than O.D.)	Nasal membranes congested at times. Post-nasal drip.		Evidence of antrum infection in past.	O.S. tends to be higher than O.D.	Same O.U. Occasional enlargement of blind spots.	O.S. definitely worse than O.D.	O.S. more remote than O.D.	
7 CCC 68	O.S.	O.S.	Bilateral sinusitis (antra). (Craig) Blockage left nostril, left ethmoiditis. (Slack) Pain O.S. associated with swelling of left turbinate. Turbinates pressed against septum, especially left side.	Pathogenic S. aureus in nose.		Usually higher in O.S.	O.S. worse than O.D.	O.S. usually poorer than O.D. when not the same O.U.	O.D. slightly more remote than O.S.	

* The term, pathogenic, as used in this survey refers to streptococci and staphylococci which have reacted positively to *in vitro* tests for pathogenicity. These tests have been used because of their close correlation with *in vivo* pathogenicity tests as determined by animal inoculation. [5]

The presence of coliform bacteria in the nasal membranes is considered a pathologic finding because of the pathogenicity of these organisms for animals.[7]

The presence of streptococci in the nasal membranes, whether pathogenic by *in vitro* tests or not, is abnormal and has been found to be associated with pathologic conditions. [8]

† Key to the abbreviations used in the table: O.D., right eye; O.S., left eye; O.U., both eyes; Staph., staplylococci; Strep., streptococci; L.P., light perception; H.M., hand movements; P.H., pinhole; cc, with correction; sc, without correction.

TABLE I.—Continued

Survey of 53 Unselected Cases of Chronic Simple Glaucoma in Relation to Nasal Pathology.

PATIENT AGE	SITE OF GLAUCOMA	EYE MOST AFFECTED	SINUSES			TENSION	FIELDS	MAXIMUM VISUAL ACUITY	NEAR POINT OF ACCOMMODATION	REMARKS
			EXAMINATION	BACTERIOLOGY	ROENTGEN RAY					
8 EB 58	O.U.	O.U.	Nasal membranes dry and crusted, left more than right.	No pathogens recovered from left nostril. Coliform bacteria in right.		Approximately the same O.U.	Approximately the same, O.D. slightly worse at times.	Same O.U.	Same O.U.	Cupping of right disk.
9 SB 53	O.U.	O.S.	Recurrent sinus attacks.	Strep. in left nostril. Coliform bacteria in nasopharynx. Later pathogenic Staph. in left nostril.	Left frontal cloudy. Both ethmoids cloudy, left more than right. Both antra cloudy.	O.S. usually higher than O.D.	Approximately the same.	Same O.U.	Same O.U.	Mild vascular change O.U. Discs normal.
10 WA 65	O.U.	O.D.	Right middle turbinate more congested than left.	Pathogenic Staph. in both nostrils.	Chronic sinusitis right frontal, both ethmoids, both antra especially marked in right. (Law).	Consistently higher O.D.	O.D. worse than O.S.	Markedly poorer O.D.	Much more remote O.D.	Deep cupping O.U.
11 HEA (Age not given)	O.U.	O.D.	Post-nasal discharge. Tonsil on right a possible focus also.	Coliform bacteria in both nostrils.	Apparently negative. (I. Schwartz).	About the same O.U.	O.D. worse than O.S.	O.D. poorer than O.S. until cataract formation O.S.	Progressively more remote O.D. O.S. remained approximately the same until cataract formation.	Cupping and atrophy of nerves O.U. O.D. worse than O.S.
12 KC 43	O.U.	O.D.	Chronic rhinitis and muco-purulent discharge, congested pharynx. Both antra cloudy, especially left. Sinus considered focus of infection by Dr. Palmer.			O.D. slightly higher than O.S.	O.D. worse than O.S.	O.D. markedly poorer than O.S.	O.D. vision too poor for measurement. O.S. 160/1M.	
13 JB 60	O.U.	O.U.	Chronic post-nasal discharge. Bilateral nasal obstruction.			About the same O.U.	Approximately the same. Improved to normal 3 months after first observation.	Same O.U.	Same O.U.	
14 HCD (Age not given)	O.U.	O.D.	Chronic post-nasal catarrh. (Richards).		Left sphenoid slightly cloudy, Ethmoiditis both sides, right more than left. Both antra cloudy, right more than left. (Feinman).	About the same O.U.	O.D. definitely worse than O.S.	Same O.U.	Same O.U.	Eye improvement associated with nasal improvement.
15 GM 63	O.U.	O.D.			Chronic sinusitis involving both maxillary and ethmoidal sinuses.	O.D. always higher than O.S.	O.D.—No L. P. O.S.—Restricted.	O.D.—No L. P. O.S.—Poor.	Vision too poor for measurement.	

TABLE I.—Continued

Survey of 53 Unselected Cases of Chronic Simple Glaucoma in Relation to Nasal Pathology.

PATIENT AGE	SITE OF GLAUCOMA	EYE MOST AFFECTED	SINUSES			TENSION	FIELDS	MAXIMUM VISUAL ACUITY	NEAR POINT OF ACCOMMODATION	REMARKS
			EXAMINATION	BACTERIOLOGY	ROENTGEN RAY					
16 LR 44	O.U.	O.D.	Possible chronic sinus trouble. (Faulkner).		Both antra cloudy, especially right. Chronic catarrhal type of infection. (C. Schwartz).	O.D. markedly and consistently higher than O.S.	O.D. more restricted than O.S. Improved and remained stationary after nasal treatment.	Approximately the same O.U.	Same O.U.	Blind spots improved with nasal treatment.
17 WR 40	O.U.	O.D.	History of sinusitis, worse on right side, 3 years.			O.D. markedly higher than O.S. until filtering operation after which O.S. was higher.	O.D. much worse than O.S.	O.D. much poorer than O.S. Vision O.D. finally reduced to H.M.	O.D. — Vision too poor for measurement. O.S.—Improved from 260/400 to 180/400.	Deep cup O.D. No cup O.S., but nerve congested.
18 FG 58	O.U.	O.D.	Recurrent blockage of right nostril. Large middle turbinate, right turbinate cauterized. Puncture of right antrum. (Ryan).	Pathogenic Staph. both nostrils.	No evidence of sinus disease. (Doctors Hospital, N. Y.).	Consistently higher O.D.	O.D. much worse than O.S.	Normal O.U.	Same O.U.	Deep cupping O.D. Slight cupping O.S.
19 KG 43	O.U.	O.U.	Sinusitis, pus in adnoids. (Kernan).		Chronic bilateral ethmoiditis. Occasional infection of antra. (C. Schwartz)	Approximately the same O.U.	Approximately the same O.U.	Normal O.U.	Same O.U. Approximately normal.	Pain O.U. definitely associated with nasal exacerbations.
20 ABG 62	O.S.	O.S.	History of infection of sinuses for 10 yrs., especially in left antrum. Involvement of left sphenoid and ethmoid. (Babcock).	Coliform bacteria in both nostrils.	Atrophic type of sinusitis, worse on left side. (Holden).	O.D.—Normal. O.S.—High.	O.D.—Normal. O.S.—Markedly contracted.	O.S.—Markedly poorer than O.D.	In contrast to other findings, O.D. more remote than O.S.	Shallow cupping O.S. O.D. normal.
21 WSH (Age not given)	O.U.	O.D.	High deflected septum to right. Middle turbinates inflamed, especially right. Chronic ethmoiditis over long period. (Hirst).	Pathogenic Staph. in both nostrils.	No active infection. (C. Schwartz). Right ethmoid shows involvement.	O.D. usually higher than O.S.	O.D. worse than O.S.	About the same O.U., with tendency to be poorer O.D.	Same O.U.	Right ethmoiditis at time of highest tension recorded.
22 HH 60	O.S.	O.S.	Left middle turbinate dry and crusted.	*Left Nostril*— Coliform Bacteria. *Right Nostril*— Pathogenic Staph.	Antrum infection from dental region, 12 years, left more than right.	O.D. 21 Only one O.S. 60 observation	Enlarged blind spot, nasal depression O.S. O.D. normal.	O.D. cc 20/30 O.S. cc 20/200.	Not taken.	Acute attack of glaucoma O. S. following activation of left antrum infection for 10 days. Glaucoma and antrum infection both of 12 years' duration. O.D. never affected.

TABLE I.—Continued

Survey of 53 Unselected Cases of Chronic Simple Glaucoma in Relation to Nasal Pathology.

PATIENT AGE	SITE OF GLAUCOMA	EYE MOST AFFECTED	SINUSES			TENSION	FIELDS	MAXIMUM VISUAL ACUITY	NEAR POINT OF ACCOMMODATION	REMARKS
			EXAMINATION	BACTERIOLOGY	ROENTGEN RAY					
23 EH 67	O.U.	O.D.	Chronic rhinitis treated by rhinologist. Post-nasal drip. Marked pharyngeal band right side. Right middle turbinate larger. Chronic blockage and inflammation of right nostril.	*Right Nostril*— 1941 Pathogenic Strep. 1942 Pathogenic Strep. *Left Nostril*— 1941 Non-pathogenic Strep. 1942 Non-pathogenic Staph.		O.D. usually higher until filtering operation.	Approximately the same O.U.	Poorer O.D.	Same O.U.	Acute attack O.D. following cold in which secretion on right side was more profuse than left. Always a definite association between nasal condition and pain in eye.
24 LJ (Age unknown, 65?)	O.U.	O.D.	Marked involvement both antra, slight involvement of ethmoids, chronic left frontal. (Ramirez).	Pathogenic Staph. in both nostrils.		Consistently higher O.D.	O.D. much worse than O.S.	O.D.—H.M. O.S. cc ranged between 20/30 and 20/100.	O.D.-Vision too poor for measurement. O.S. 160/500.	Since first observation, left antrum has become more involved and fields O.S. have contracted progressively.
25 AK 59	O.U.	O.U.	Considerable deflection of septum. Middle turbinates pressed against septum. "Catarrhal nose."	Pathogenic Strep. in nostrils from 1931-1939. None since then.		Same O.U.	Same O.U. Improved to normal.	Same O.U.	Approximately the same O.U.	With vaccine therapy there was improvement in clinical symptoms of focal infection and fields. Bacteriology of nostrils became normal and tension was more easily controlled.
26 MK 49	O.U.	O.S.	History of sinusitis for 12 years, especially left antrum. Submucous operation by Dr. Craig.			Same O.U. at first. Later O.S. higher than O.D.	Approximately the same O.U.	Same O.U.	Approximately the same O.U.	Eye symptoms improved after submucous operation, reduction of vascular hypertension and treatment of chronic colitis.
27 KML (Age not given)	O.U.	O.D.	Sinus infection right side for 3-4 years, right side operated upon. Deflected septum high right.	Non-pathogenic Staph. in nostrils.		Same O.U.	Same O.U.	Poorer O.D.	O.D. much more remote than O.S.	Cupping and atrophy O.U., especially O.D.
28 AL 57	O.U.	O.S.	Mucoid discharge from left nostril. Septal spur on left. Ocular symptoms O.S. associated with left nasal congestion.	*Right Nostril*— Pathogenic Strep. *Left Nostril*— Pathogenic Strep. and coliform bacteria.	No evidence of active sinus infection. (C. Schwartz).	O.S. consistently higher than O.D.	Worse O.S.	Same O.U.	Approximately the same O.U.	

TABLE I.—Continued

Survey of 53 Unselected Cases of Chronic Simple Glaucoma in Relation to Nasal Pathology.

PATIENT AGE	SITE OF GLAUCOMA	EYE MOST AFFECTED	SINUSES			TENSION	FIELDS	MAXIMUM VISUAL ACUITY	NEAR POINT OF ACCOMMODATION	REMARKS
			EXAMINATION	BACTERIOLOGY	ROENTGEN RAY					
29 EL 29	O.U.	O.S.	Frequent colds. Nasal treatments. Left antrum darker than right. Posterior tips of inferior turbinates somewhat hypertrophied. (Bowers).	Pathogenic Staph. in nostrils. Coliform bacteria in nasopharynx. After vaccine therapy, cultures yielded no pathogenic bacteria.	Slight haziness of both ethmoids. (Bowers).	Same O.U. for past 7 years. Before that tended to be higher O.S. than O.D.	Relative central scotoma for blue O.U. when first seen. Normal O.U. now.	Poorer O.S. than O.D. at first observation. Improvement O.S. Present vision normal O.U.	Same O.U.	Labyrinthitis left. Tension has been controlled with pilocarpine 1% since starting vaccine therapy in 1936—before that, tension O.S. showed an occasional marked increase in spite of miotics.
30 MM 59	O.D.	O.D.	Occasional pain in right frontal. Pressure and redness of middle turbinates especially right. Nasal septum deflected to the right, occasional lesion on right septum and in right nostril.			O.D. higher than O.S. before miotics. Normal O.U. under 1/8% pilocarpine.	At first O.D. worse than O.S. Improved to normal O.U.	Same O.U.	Same O.U.	Fields improved after intestinal treatments. Pain O.D. disappeared after nasal treatments.
31 EM 45	O.U.	O.S.	Left sinuses always more troublesome than right.	Coliform bacteria and pathogenic Strep. in nasopharynx.	Sphenoid sinuses multilocular. One posterior median locule cloudy. (I. Schwartz).	O.S. usually higher than O.D.	O.S. much worse than O.D.	O.S. poorer than O.D.	Same at first. Later O.S. became more remote.	Cupping O.S. always more marked than O.D. Both eyes operated upon—much slower recovery O.S. Nasal exacerbation at same time. Eye inflammation and irritation did not subside until nasal condition improved.
32 SM 39	O.U.	O.S.	Nasal membranes dry, otherwise normal.	*Left Nostril*—Coliform bacteria. *Right Nostril*—Pathogenic Staph.		O.S. usually higher than O.D.	Same O.U. at first Later improved to normal.	O.S. much poorer at first. Later improved to normal O.U.	O.S. definitely more remote at first. Later O.U. improved to approximately the same.	Nasal bacteriology worse on left side only indication of nasal pathology. O.S. more painful and congested than O.D.
33 JM 68	O.U.	O.S. (Formerly O.D.)	Naso-pharynx slightly congested. Otherwise normal.	Coliform bacteria in both nostrils.		O.S. consistently higher than O.D. since observation by us.	O.D. worse than O.S. (Previous glaucoma?) O.S. progressively worse.	O.D. worse than O.S. (Previous glaucoma?) O.S. progressively poorer.	O.D. more remote than O.S. (Previous glaucoma?).	Glaucoma O.D. 4 years prior to observation by us. Trephine O.D. 3 years previously. Glaucoma O.S. now active.

TABLE I.—Continued

Survey of 53 Unselected Cases of Chronic Simple Glaucoma in Relation to Nasal Pathology.

PATIENT AGE	SITE OF GLAUCOMA	EYE MOST AFFECTED	SINUSES			TENSION	FIELDS	MAXIMUM VISUAL ACUITY	NEAR POINT OF ACCOMMODATION	REMARKS
			EXAMINATION	BACTERIOLOGY	ROENTGEN RAY					
34 GO 49	O.U.	O.D.	Frequent sinusitis—chronic marked post-nasal discharge. Narrow poorly spaced nose with deflected and thickened septum—poor ventilation and drainage of both ethmoids. (Evans).		No evidence of infection. (Midtown Hosp., N. Y.) Negative (Evans).	Same O.U. except occasionally O.D. higher than O.S.	O.D. markedly worse than O.S. at first. Both fields improved but O.D. still worse.	Improved from 20/40 O.U. cc to 20/15 O.U. sc after vaccine therapy.	Same O.U.	
35 LP 60	O.U.	O.S. (Slightly more)	History of serious sinus trouble—operations. Chronic bilateral ethmoiditis—believe sinusitis to be source of eye trouble. (Faulkner). Frequent sinus attacks—left antrum, sphenoid and ethmoid sinuses always worse.		Frontals only fairly clear. Thickening in ethmoids and antra; sphenoid sinuses rather cloudy. (Dixon).	About equal O.U. throughout.	Normal O.U. at first. Later O.S. markedly contracted at same time as left sinuses were filled with pus.	Considerable variation O.U. Between 20/15 cc and 20/40 cc.	Same O.U.	After operation on ethmoids and sphenoids, tension was reduced and successfully controlled by miotics. Tension O.S. increased at time of left sinusitis (ethmoid and sphenoid).
36 LP 49	O.U.	O.D.	Chronic sinusitis. (Craig). Condition worse on right side.		Evidence of old sinus infection. Bilateral ethmoiditis. (Schwartz).	Approximately the same O.U.	O.D. worse than O.S. throughout.	O.D. poorer at first. Later improved to approximately the same O.U.	O.D. slightly more remote than O.S.	Infected tissue at base of right tonsil undoubtedly a factor in her (eye) condition. (Craig).
37 MP 46	O.U.	O.S.	Nasal congestion associated with pain in left eye.	Pathogenic Staph. in both nostrils.	Left antrum smaller than right, shows slight cloudiness. Suggestive of old infection. (N. Y. Hosp.).	Tendency for O.S. to go beyond normal limits. Higher than O.D.	Same O.U.	Same O.U.	O.S. more remote than O.D. at first. Later improved to approximately the same O.U.	Cupping O.S. definitely worse than O.D.
38 BR 52	O.D.	O.D.	Sinus trouble for 10 years. Nasal obstruction by deflected septum to right. Hypertrophy of all turbinates, especially right. Chronic suppuration of nasal accessory sinuses. (Zwerling). Right ethmoid hypertrophic. (Zwerling). Frequent sinus attacks, especially right antrum. Chronic blockage of right nostril.	B. coli in nostrils and naso-pharynx.	Involvement of post-ethmoids right side. (Levy).	O.D. consistently higher than O.S. until operation. Still tends to be higher especially early a.m.	O.D. definitely worse than O.S.	Corrected vision the same O.U. at first but P.H. vision O.D. poorer than O.S. Later vision O.D. much poorer than O.S. Lens O.D. cataractous.	Same O.U.	Iridocorneosclerectomy O.D.

TABLE I.—Continued

Survey of 53 Unselected Cases of Chronic Simple Glaucoma in Relation to Nasal Pathology

PATIENT AGE	SITE OF GLAUCOMA	EYE MOST AFFECTED	SINUSES			TENSION	FIELDS	MAXIMUM VISUAL ACUITY	NEAR POINT OF ACCOMMODATION	REMARKS
			EXAMINATION	BACTERIOLOGY	ROENTGEN RAY					
39 ER 73	O.U.	O.S. (O.D. previously)	History: Involvement of right posterior ethmoids and sphenoids. Four operations. Observation here: At this time O.S. more involved. Considerable post-nasal discharge especially left. Maxillary sinuses cloudy on left. Much purulent discharge from antrum; general involvement of frontals and ethmoids. (Sauter).	Pathogenic Staph. in both nostrils.		O.D. higher than O.S. in spite of stronger miotics O.D. No drainage operation on O.D.	O.D.—faulty light projection. O.S.—Marked contraction.	O.D.–None for three years before observation here.	O.D.—No L.P. O.S.—Slightly subnormal.	
40 ER 67	O.S. O.D. (?)	O.S.	History of marked chronic inflammation both antra, with polypoid masses. Radical operation on antra and ethmoids. (Abraham). 1938 — Both antra densely cloudy, almost opaque. (Phillips). 1942—Antra densely cloudy Sphenoid sinuses cloudy. Ethmoids densely cloudy or opaque, left worse than when last seen.			O.S. higher than O.D.	O.S. definitely more contracted than O.D.	Normal O.U.	Same O.U. at first, later O.S. more remote than O.D.	Deep cupping O.S. Physiologic cupping O.D.
41 VS 50	O.U.	O.D.	Right sided rhinitis. Chronic sinusitis over period of 20 years, right more than left. Right middle turbinate pressed against septum. Constant crusting and redness of right middle turbinate and septum.	Pathogenic Staph. in noth nostrils.		O.D. usually higher than O.S. until filtering operation O.D. O.S. rose suddenly at one time and operated upon. Tendency still for O.D. to be higher than O.S.	Same O.U. at first. Later O.D. markedly worse than O.S.	O.D. poorer than O.S. at first. Later O.S. developed cataract, vision poor.	Same O.U. at first. Later O.D. more remote than O.S.	Repeated attacks of right sided rhinitis associated with glaucomatous attacks O.D.
42 ES 64	O.D. O.S. (?)	O.D.	Clinically not suspicious except for considerable post-nasal secretion.	Coliform bacteria in both nostrils and in naso-pharynx.		O.D. tends to be higher than O.S. but both within normal limits.	O.D. definitely glaucomatous. O. S. normal.	Same O.U. at first, later O.D. poorer than O.S.	O.D. more remote than O.S. After operation on O.D., O.S. was more remote.	Cupping O.D.

TABLE I.—Continued
Survey of 53 Unselected Cases of Chronic Simple Glaucoma in Relation to Nasal Pathology

PATIENT AGE	SITE OF GLAUCOMA	EYE MOST AFFECTED	SINUSES			TENSION	FIELDS	MAXIMUM VISUAL ACUITY	NEAR POINT OF ACCOMMODATION	REMARKS
			EXAMINATION	BACTERIOLOGY	ROENTGEN RAY					
43 LS 40	O.U.	O.S.	Dr. Saunders removed pus and polypi from left ethmoidal sinus, opened sphenoid, found pus. Later radical antrum left side, left frontal blocked. Chronic hyperplastic sinusitis of all sinuses.	No pathogenic organisms in nostrils. Cultures taken immediately after sulfonamide therapy.	Infection of left frontals and ethmoids, both antra. (Johns Hopkins Hosp.)	Usually higher in O.S. especially during sinus attacks.	O.S. more contracted than O.D. at first. Later about the same O.U.	O.D. poorer than O.S. due to corneal scars. Vision O.S. improved with nasal improvement.	O.D. more remote than O.S.	Nerve O.S. paler than O.D. with definite cupping. Infected tooth found to extend into left antrum, Dr. Saunders of the opinion that patient's eye condition bears a direct relationship to condition of frontal sinus. Rheumatoid arthritis of spine and myositis of left shoulder both on basis of focal infection. (Pierce).
44 NS 63	O.U.	O.D.	Marked deflection of nasal septum to right, mucous membrane of entire nose thickened, also lower and middle turbinates. Right ethmoiditis.	Pathogenic Staph. in both nostrils.	Right ethmoid very cloudy, black, left slightly cloudy.	Same O.U. at first. Since filtering operations O.U., O.S. tends to be higher.	O.D. definitely worse than O.S.	O.D. consistently and markedly much poorer than O.S. Improved after sinus treatment but later became worse again.	O.D. much more remote than O.S.	Pus in tonsils, especially right.
45 HAS 68	O.U.	O.D. (Slightly more than O.S.)	High deflection of septum to left, hypertrophy of middle turbinate. (Perrault).	Pathogenic Staph. in both nostrils.	Anatomic variation involves primarily right frontal, but greater opacity in left ethmoid and maxillary structures. (St. Lukes Hosp., N. Y.).	O.D. higher than O.S. before filtering operations, but low O.U. since then.	Same O.U. at first. Later O.D. much worse.	Same O.U. at first. Later O.D. definitely poorer than O.S. Cataracts developed O.U.	Same O.U. at first but later O.D. had only light perception.	Cupping O.U.
46 CS 63	O.S.	O.S.	History of acute maxillary sinusitis, left. Septum deviated to right. Pain in left cheek-bone when patient gets a cold.			O.S. considerably higher than O.D. Uncontrolled at times when nasal condition was bad.	O.D.—Cataract. O.S.—Somewhat contracted at first. Improved to normal.	Faulty light perception in O.D. due to retinal hemorrhage O.D., associated with phlebitis in right leg. O.S. 20/30 ee except during nasal involvement when vision became poorer temporarily.	O.D.—Vision too poor for measurement. O.S. 270/300 at first observation. 110/300 when last seen	
47 JT 61	O.U.	O.S. (Slightly more than O.D.)	No secretion or obstructions, nasal mucosa not congested. (Woodruff). Later pan-sinusitis attack, involving antra and ethmoids.		Chronic sinusitis involving both ethmoids, right antrum. Posterior ethmoid cells most involved. (Law).	O.S. higher than O.D. when first observed but later approximately same O.U. with occasional rise in O.D.	Same O.U.	O.S. usually poorer than O.D., but improved to normal O.U.	O.S. more remote at first. Later improved. Now same O.U.	

TABLE I.—Continued

Survey of 53 Unselected Cases of Chronic Simple Glaucoma in Relation to Nasal Pathology

PATIENT AGE	SITE OF GLAUCOMA	EYE MOST AFFECTED	SINUSES			TENSION	FIELDS	MAXIMUM VISUAL ACUITY	NEAR POINT OF ACCOMMODATION	REMARKS
			EXAMINATION	BACTERIOLOGY	ROENTGEN RAY					
48 CT 48	O.U.	O.U.	History of turbinate infection, necessitating cauterization. Low grade sinus condition with post-nasal drip, — treated. Cloudiness of all sinuses. (McCollough).			Approximately the same O.U.	Same O.U. (Normal at all times).	Normal O.U.	Same O.U.	Pan-sinusitis (chronic) and bilateral glaucoma. Sinusitis and vision improved.
49 HW 66	O.U.	O.S.	Muco-pus in left middle turbinate.	Pathogenic Strep. and Staph. in both nostrils. Pathogenic Strep. from nose produced iritis when injected intravenously into rabbits.		O.S. always higher than O.D., even when tension O.U. was within normal limits.	O.S. more contracted than O.D.	O.S. better than L.D. in spite of increased tension O.S. Vision O.U. became very poor during attack of sinusitis but improved when sinuses did.	O.S. more remote than O.D. at first. Later the same O.U.	Retinal hemorrhage O.D. after cold involving sinuses.
50 PW 60	O.U.	O.D.	Right middle turbinate often red and crusted. Right nostril blocked when patient has cold.	Many diphtheroids in nostrils. Subsequent cultures revealed no pathogenic organisms in nostrils.		Provocative tests did not increase tension beyond 25 mm. Hg. (Schiotz) O.U., but under emotional strain, it rose as high as 38 mm. Hg. (Schiotz) O.U.	O.D. slightly worse than O.S.	Same O.U. at first. Later O.D. poorer than O.S.	Approximately the same O.U.	Cupping O.D. No cupping O.S.
51 UYY 54	O.U.	O.S.	Chronic post-nasal drip and secretion in nose.	Pathogenic Staph. in both nostrils.	Mild low-grade chronic ethmoiditis both sides, most marked in posterior cells, of hyperplastic type. (Law).	Never above 25 mm. Hg. (Schiotz) in either eye.	Slightly worse O.S.	Slightly poorer O.S.	Somewhat more remote O.S.	Cupping O.U., more marked O.S.
52 MY 30	O.U.	O.S. (Slightly)	"Bad sinuses, recurrent headaches for 15 years." (Patient). Septum deviated to right. Left antrum cloudy, left middle turbinate edematous, pus from left sphenoid. (Weymuller).	Pathogenic Staph. in both nostrils.	Left antrum cloudy, left sphenoid small. Both sphenoids cloudy. (I. Schwartz).	At first same O.U. Since filtering operations O.U., tension O.S. has always been higher than O.D.	Markedly worse O.S.	Always poorer O.S. than O.D.	O.S.–Vision too poor for measurement. O.D.—190/3M.	Cupping O.U. Patient notices definite association between nasal and ocular symptoms.
53 RG 50	O.U.	O.D.	Chronic nasal blockage.	Pathogenic Strep. in both nostrils. Pathogenic Staph. in left nostril.	Right ethmoid cloudy. Definite increase in density of right antrum. Both frontals cloudy.	O.D. consistently higher than O.S.	O.D. definitely worse than O.S.	Same O.U.	O.D more remote than O.S.	

cant. However, in three instances the presence of pathogenic organisms indicated nasal pathology, although nasal examination and roentgenographic examination revealed normal findings. Coliform bacteria were the organisms recovered in each instance.

With the exception of three cases, pathogenic organisms were recovered from the nasal membranes of each patient studied bacteriologically. In the first of the three instances, the cultures were taken immediately after sulfonamide therapy had been administered which may have accounted for the negative findings. In the second, there were many diphtheroid bacilli of unknown pathogenicity, and in the third, there was unquestionable pathology in the right sinuses although nasal cultures yielded no pathogens.

or mixed cultures. Since nasal infection is commonly attributed to toxic staphylococci, the presence of other pathogens in 55 per cent of these cases may be of some significance. However, we do not believe that cultures from the nasal membranes tell the whole story of sinus infection, since the organisms present in the posterior sinuses are to be found in the postnasal secretion; consequently they cannot be differentiated from those harbored in the throat.

In short, it would appear that bacteriologic investigation of the nasal membranes may be a useful adjunct to nasal examination and roentgenographic examination of the sinuses. However, direct culture from the affected sinuses would undoubtedly prove more enlightening and perhaps reveal hidden pathology in certain cases.

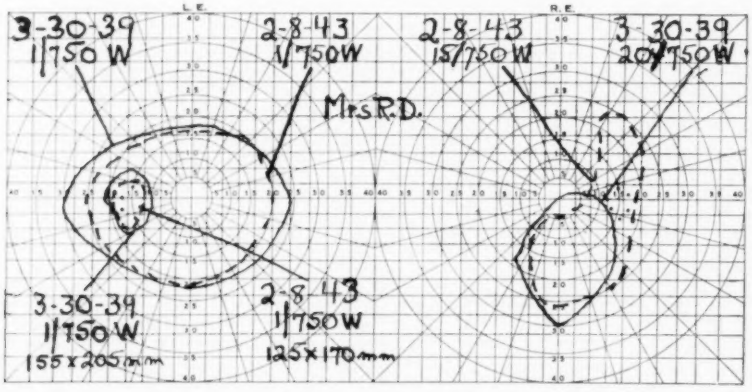

FIG. 1.—Visual fields in case 1 (Mrs. R. D.), chronic simple glaucoma, O. D. and O. S., acute glaucoma O. D. and O. S. Unbroken line represents fields taken on 3/30/39; O. D. with a 20 mm. white test object at 750 mm. on a neutral gray tangent screen with 7-foot candles of artificial daylight illumination (Macbeth). O. S. with a 1 mm. spherical white test object at 750 mm. The broken lines represent the fields taken on the right and left eyes on 2/8/43. O. D. with 20 mm. white test object; O. S. with 1 mm. white test object, neutral gray tangent screen with 7-foot candles of artificial daylight illumination at 750 mm.

Of the 31 cases in which pathogens were recovered from nasal cultures, 14 (45 per cent) yielded toxic staphylococci only, 8 (25 per cent) revealed streptococci in either pure or mixed culture, and 10 (30 per cent) showed the presence of coliform bacteria in either pure

The following cases are presented in detail to illustrate findings typical of those summarized in table I.

Case 1—Chronic simple glaucoma, O. D., followed by an attack of acute glaucoma O. D. and O. S. Mrs. R. D., aged 39, was first examined by one of us on March 3, 1939. She gave a history of having noticed a "film"

over her right eye during the past year, accompanied by pain and decreasing vision. Two weeks prior to our observation of her, she was told by her physician that she had acute glaucoma (tension 90 mm. Hg. O. D.) and a 2 per cent solution of pilocarpine was prescribed. This reduced but did not control tension, and her ocular symptoms were not alleviated. Her history also included a 75 per cent loss of hearing in the *right* ear, reported by Dr. C. J. Imperatori in 1933, and roentgenograms taken at that time by Dr. F. M. Law showed a mild chronic ethmoiditis on both sides, extending into the left antrum, with evidence of a mild chronic process in the *right* mastoid.

Nasal examination at this time showed the septum markedly deflected to the *right* and postnasal discharge, but the nasal membranes appeared to be fairly normal.

A 2 per cent solution of pilocarpine, instilled five times a day in the right eye, did not control the tension and on April 19, 1939, an iridocorneosclerectomy was performed. Since that time tension in the right eye has remained within normal limits with the use of miotics. Three days after surgery on the right eye the patient developed acute glaucoma in the left eye which was successfully controlled by miotics.

In June of 1939, Dr. G. G. Boyd reported that pus was being recovered from the pa-

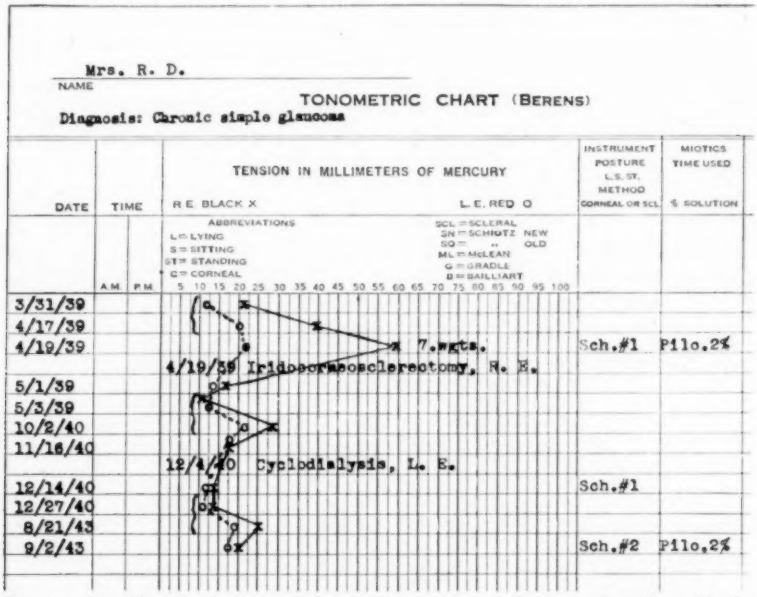

FIG. 2 — An abbreviated graph of the tension in case 1. The unbroken line with the crosses indicates the right eye. The graph shows the tension in the right eye remained higher over a period of four years in spite of an iridocorneosclerectomy performed on the right eye.

Her corrected vision was found to be 20/200 in the right eye and 20/20-1 in the left eye; there was no improvement with correction. Fundus examination revealed deep cupping of the right optic nerve, none of the left. The tension was 56 mm. Hg. (Schiötz), O.D. 14 mm. Hg. (Schiötz), O.S. Slit lamp examination revealed edema of the right cornea, but no keratic precipitates or cells in the anterior chamber and no aqueous beam. Near point of accommodation was 270 mm. for 3 meter print, O.D., and 220 mm. for 400 mm. print, O. S. The visual fields were as shown in figure 1 (unbroken line).

tient's *left* sphenoids, and in August of the same year the patient was troubled with pain and blurring of vision in the *left* eye. Nasal treatment with improvement in the sinusitis was accompanied by improvement of the ocular symptoms. In May, 1940, the left eye again became troublesome after an attack of sinusitis on the left side and became progressively worse until in December, 1940, it was considered advisable to operate. A cyclodialysis was performed. At this time Doctor Boyd reported that mucopus was present in the left posterior ethmoidal region and nasopharynx. Tension in the left eye has remained consis-

tently within normal limits except during the attack of acute glaucoma following sclerectomy on the right eye. The visual field of the left eye remained normal until the early part of 1943 when a slight contraction was noted.

On April 28, 1943, the visual fields were as shown in figure 1 (broken line).

Following sclerectomy on the right eye, tension has remained within normal limits and approximately the same in both eyes, with a slight tendency to be higher in the right eye (fig. 2). The near point of accommodation has improved to 250 mm. for 2 meter print in the right eye and to 160 mm. for 300 mm. print in the left eye. Vision in the right eye has improved to 20/70 with correction and in the left eye to 20/20 with correction, except during exacerbation of sinus disease when vision was temporarily impaired in both eyes.

It has been necessary for the patient to remain under the constant care of a rhinologist because of recurrent attacks of sinusitis which are usually much more marked on the right side. Exacerbations of the sinus condition have invariably been associated with increased ocular symptoms. Nasal cultures in 1941 showed the presence of streptococci.*

Discussion of Case 1: This patient apparently had chronic simple glaucoma in both eyes and, in the progress of the disease, developed attacks of acute glaucoma. The eye most seriously involved in the glaucomatous process was the right eye and the involvement of the sinuses on the right side was greater than on the left. Exacerbations of increased tension almost invariably coincided with exacerbation in the severity of the sinus infection, and surgery on each eye did not control tension without miotics.

Case 2 — Chronic simple glaucoma, O. D., and right posterior ethmoiditis and sphenoiditis. Mrs. B. R., aged 52, was first examined on January 28, 1939. She gave a history of an attack of "fogginess" of vision which had occurred the previous year and which had been considered unimportant. During another attack one month before our observation, tension was found to be 57 mm. Hg. (Schiötz) in the right eye and 28 mm. Hg. (Schiötz) in the left eye. A 1 per cent solution of pilocarpine failed to control tension, but pilocarpine, eserine and dionin reduced it to 28 mm. Hg. (Schiötz) in the right eye, and to 20 mm. Hg. (Schiötz) in the left eye. Visual fields were not recorded. The patient also reported having had four episodes of loss of vision in the right eye, the first occurring three months before examination here. Pain or redness were not associated with these attacks. There was a history of recurring attacks of sinusitis during the past 10 years.

When first seen by us, 15 hours following the use of pilocarpine, eserine and dionin, the tension was 17 mm. Hg. (Schiötz) in the right eye and 11 mm. Hg. (Schiötz) in the left eye. Corrected vision was 20/40 in each eye, but pinhole vision was 20/40 in the right

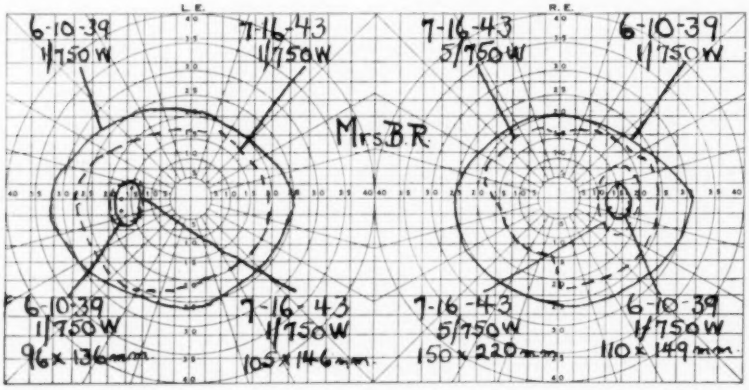

FIG. 3 — Visual fields in case 2 (Mrs. B. R.), chronic simple glaucoma, O. D. Unbroken line, 6/10/39, represents the field of the right eye taken with a 1 mm. white test object at 750 mm. on a neutral gray tangent screen with 7-foot candles of artificial daylight illumination. The unbroken line in the left eye was also taken with the same size test object and under the same conditions. Broken lines, 7/16/43, represent the field in the right eye taken with a 5 mm. spherical white test object and in the left eye taken with a 1 mm. white spherical test object; other conditions were standardized as on 6/10/39.

* See footnote to table I.

eye and 20/25 in the left eye. Near point of accommodation was 140 mm. for 300 mm. print in the right eye, 175 mm. for 300 mm. print in the left eye. Both anterior chambers were shallow.

Visual fields were contracted as shown in figure 3 (unbroken line).

Nasal examination by Dr. S. Zwerling revealed obstruction by a deflection of the septum to the right with a ridge. There was moderate hypertrophy of all the turbinates, especially on the right side. There was also chronic suppuration of the nasal accessory sinuses.

On February 23, 1940, an iridocorneosclerectomy was performed on the right eye.

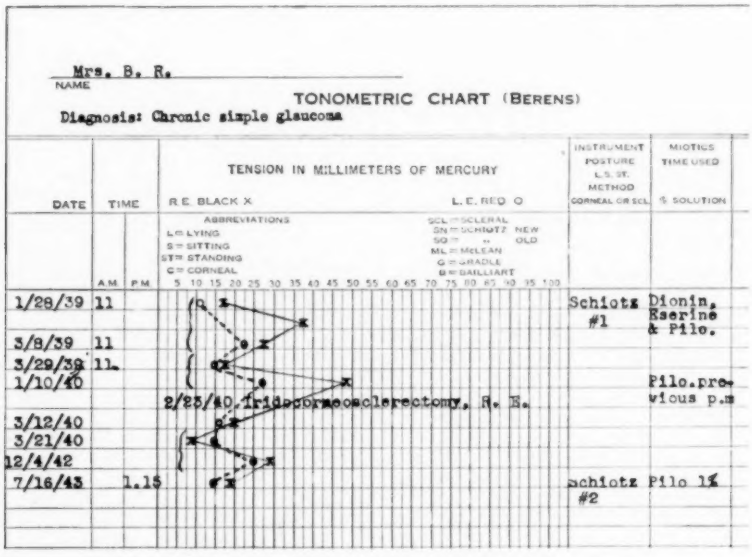

FIG. 4 — An abbreviated tonometric chart of the tension as recorded in case 2. The broken line and crosses indicate the tension in the right eye and show that tension was persistently higher in the right eye in spite of an iridocorneosclerectomy performed on this eye.

Before operation tension in the right eye was consistently higher than in the left, but after that, was approximately the same in both eyes up to the beginning of 1943 (fig. 4). Since then tension in the right eye has again been higher than in the left eye, though within normal limits. Vision has been variable; in the right eye it has progressively diminished in spite of a good filtering cicatrix. During a sinus attack in February, 1939, vision in the right eye was reduced to 10/200, but improved gradually to 20/25 during successful treatment of the sinus condition. After sclerectomy the vision was reduced to 10/200 and has not improved since. This is undoubtedly due in part to the formation of a cataract three months after operation. On the other hand, since recovery from the sinus attack in February, 1939, corrected vision in the left eye improved steadily to 20/20, and this vision has since been retained. Near point of accommodation in the left eye was 190 mm. for 400 mm. print when last tested.

Visual field changes were as shown in figure 3 (broken line).

In 1939 and 1940 pathogenic bacteria were not found in the nasal passages but repeated cultures from the nasopharynx revealed increasingly large numbers of toxic streptococci. In 1941 coliform bacteria were recovered by culture from both nostrils* and from the nasophaynx in addition to the toxic streptococci. The patient was given injections of coliform vaccine for somewhat over a year, and cultures at the end of that time failed to yield coliform bacteria in either nostril or nasopharynx. However, there were toxic streptococci in the nose* and large numbers of toxic streptococci and staphylococci in the nasopharynx.

Discussion of Case 2: Up to the present, the right nostril has been consistently more inflamed and congested than the left. There have been frequent recurrent attacks of sin-

* See footnote to table I.

usitis, especially of the right antrum. In 1941, Doctor Zwerling reported that the right ephmoid was hypertrophic. Local sinus therapy has been only temporarily beneficial, and vaccine therapy has apparently had little or no effect. Possibly it may have controlled the less active and less extensive infection of the left sinuses, as indicated by the improvement of vision in the left eye and the elimination of sinus attacks on the left side.

Apparently more radical measures are necessary for the control of the sinusitis in this case, with possibly a more extensive search for secondary foci of infection.

Case 3—Chronic simple glaucoma, O. U., condition in the right eye more advanced than in the left eye; chronic right ethmoiditis with exudate, pus in both tonsils, but the right more involved. Mrs. N. S., aged 61, was first examined by one of us on November 8, 1940. During the previous year and a half she had been under the care of an ophthalmologist who reported that her tension had been controlled by a solution of ½ per cent pilocarpine, instilled 2 to 3 times a day.

When first observed at 12 M. the tension was 25 mm. Hg. (Schiötz) in each eye, four hours after using a solution of ½ per cent pilocarpine. Corrected vision was 6/200 in the right eye and 20/20 in the left. Vision in the right eye was too poor to permit measurement of the near point of accommodation. In the left eye, it was 210 mm. for 400 mm. print. Slit lamp examination revealed no pathology in either eye. Fundus examination revealed marked temporal cupping of the right nerve, with grade I angiosclerosis. In the left eye deep temporal cupping was present, but there was no evidence of angiosclerosis. Visual fields were contracted as shown in figure 5 (unbroken line).

Roentgenograms of the nasal sinuses by Dr. H. E. Illick revealed "both ethmoid areas to be cloudy, the left only slightly so, the right moderately. Both the anterior and posterior groups of cells were cloudy due to a chronic or subacute involvement. Slight retained exudate was suspected on the right side."

Nasal examination (Dr. S. L. Craig) showed "a marked deflection of the nasal septum to the right, with thickening of the mucous membrane throughout the entire nose. There was thickening and congestion of the lower middle turbinates. A large amount of pus and cheesy material was expressed from the right tonsil, a small amount from the left. Transillumination revealed the frontal sinuses and antra to be clear. There was marked thickening in the right ethmoid region, slight in the left. Roentgenograms revealed the right ethmoid to be quite cloudy, and left slightly so."

Cultures from the nasal passages yielded a large number of pathogenic staphylococci.

Because of the advanced field changes iridocorneosclerectomy was performed on the

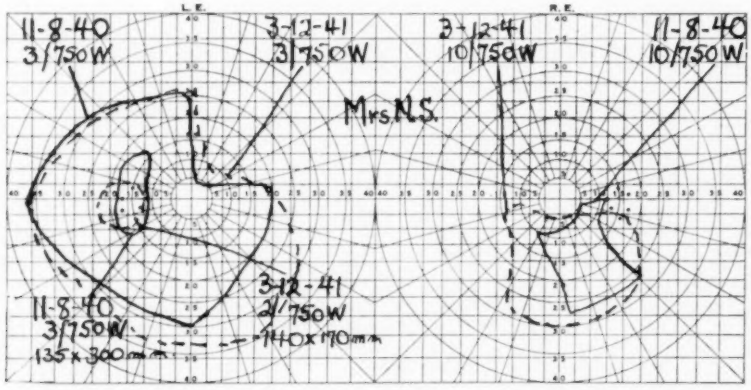

FIG. 5.—Visual fields in case 3 (Mrs. N. S.), chronic simple glaucoma, O. U. The condition in the right eye was more advanced than in the left eye. Unbroken line, 11/8/40, represents the field of the right eye taken with a 10 mm. spherical white test object at 750 mm. on a neutral gray tangent screen with 7-foot candles of illumination; the field of the left eye was taken with a 3 mm. spherical white test object and the other conditions were the same as for the right eye. Unbroken lines, 3/12/41, represent the field of the right eye taken with a 10 mm. spherical white test object and the field of the left eye taken with a 3 mm. spherical white test object. The test conditions were standardized as on 11/8/40.

right eye on January 10, 1941, and four days later cyclodialysis was performed on the left eye. A mild postoperative iritis developed in the right eye, which cleared up in three weeks. Since then tension in the right eye has ranged between 10 mm. Hg. (Schiötz) and 20 mm. Hg. (Schiötz) without the use of miotics (fig. 6). The tension in the left eye has been maintained well within normal limits by the use of miotics. There has been no great change in the near point of accommodation in either eye. Vision in the right eye improved to 20/70 after nasal treatments but subsequently decreased to 8/200. There was no appreciable change in vision of the left eye.

ethmoiditis, right antrum and both frontals involved. Mr. R. G., aged 50, was under our observation for only eleven days, but his history, coupled with our findings, was of interest in relation to this study.

A brother of the patient had been told four years previously that he had glaucoma, which made the patient apprehensive about his own eyes. Therefore, when he noticed an occasional "film" over his right eye, as well as rainbow haloes, especially when observing car lights, he insisted that his tension be taken by his physician. Because of a slight rise in tension, he was placed on pilocarpine nitrate 2 per cent three times daily, and ½ per cent

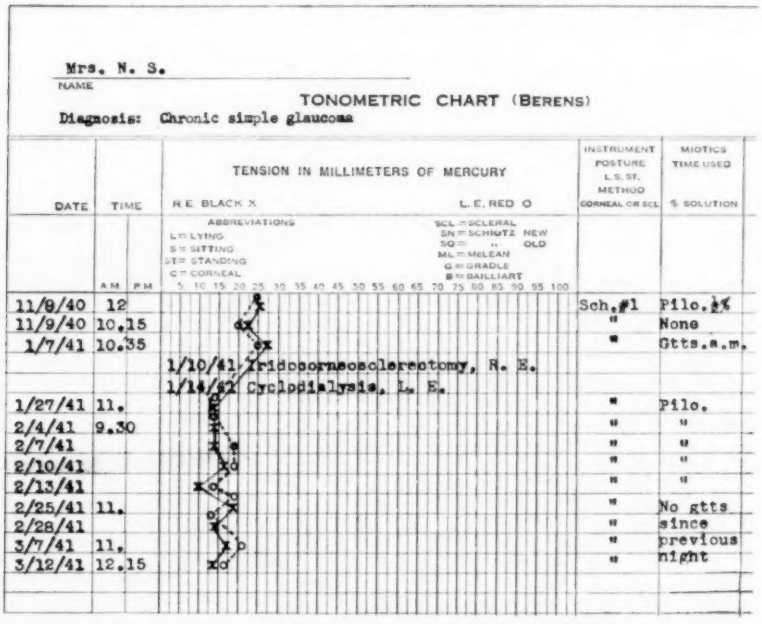

FIG. 6 — Complete tonometer chart of case 3 showing that the tension in the right and left eyes remained about the same in both eyes during the period of observation.

Visual fields taken when last observed on March 21, 1941, were as shown in figure 5 (broken line).

Discussion of Case 3: In this case the nasal pathology was unmistakably greater on the right side although both sides were involved. Glaucomatous activity, as indicated by visual field changes rather than by tension, has been markedly greater in the right eye. There was a definite association between exacerbation of the sinus condition and loss of visual acuity.

Case 4 — Chronic simple glaucoma, O. U., more advanced in right eye than in left; right

eserine in each eye at bedtime. The symptoms in his right eye disappeared and did not reoccur unless he failed to use miotics. The left eye was at all times free from these symptoms.

He was observed by us somewhat over a year after being placed on miotics. At that time his tension was 25 mm. Hg. (Schiötz) in each eye, taken at 1:30 p.m., five hours after instillation of pilocarpine 2 per cent. His uncorrected vision was 20/20-2 in the right eye and 20/20 in the left. With his reading correction, his near point of accommodation with the 400 mm. print was 250 mm. in the right eye and 240 mm. in the left. Slit lamp

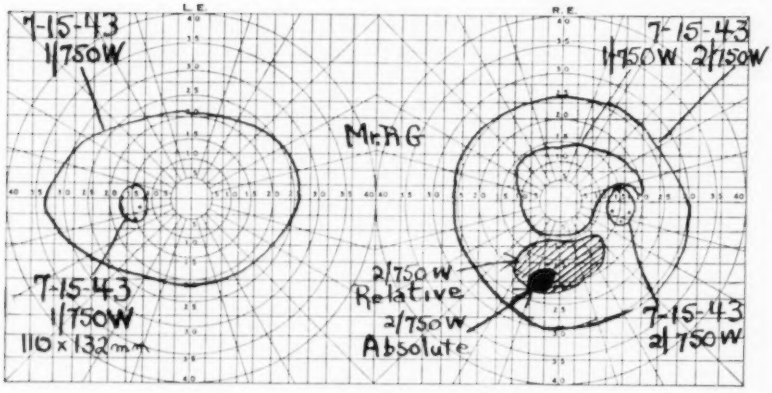

FIG. 7 — Visual fields in case 4 (Mr. R. G.), chronic simple glaucoma, O. U. The visual field of the right eye was taken with 1 and 2 mm. spherical white test objects as indicated. The field of the left eye was taken with a 1 mm. spherical white test object. The field changes were more advanced in the right eye than in the left eye. The visual field was examined at 750 mm. on a neutral gray tangent screen with 7-foot candles of artificial daylight illumination.

examination revealed no cells, no keratic precipitates and no aqueous beam in either eye. Examination with the ophthalmoscope showed cupping of the disk in the right eye, but in the left eye only a possible congestion of the nerve and no cupping. Gonioscopic examination showed the angles of both anterior chambers to be well opened, with considerable pigment over Schlemm's canal in the right eye but very little in the left. Fields were as shown in figure 7 (unbroken line).

Roentgenograms of the sinuses (fig. 8) showed cloudiness of the supra-orbital cells in the right ethmoid, definite increase in the density of the right antrum and some cloudiness in both frontals.

Bacteriologic studies of the nasal membranes revealed pathogenic streptococci* on the right side and pathogenic staphylococci on the left. Culture of nasopharyngeal secretion yielded many coliform bacteria and pathogenic streptococci.

One hour after a provocative test, in which the patient had consumed one quart of water and then been subjected to the dark room test, the tension was found to be 37 mm. Hg. (Schiötz) in the right eye and 27 mm. Hg. (Schiötz) in the left eye. The patient was placed on pilocarpine 3 per cent and was instructed to discontinue the use of eserine which we prefer to reserve for emergencies. One-half hour after a provocative test which included the use of euphthalmine, tension was 25 mm. Hg. (Schiötz) in the right eye and 21 mm. Hg. (Schiötz) in the left. The use of pilocarpine 3 per cent alone successfully maintained tension within normal limits.

FIG. 8 — Roentgenogram of sinuses in case 4 (Mr. R. G.). The plate shows definite clouding of the right ethmoid and right antrum as well as the right frontal sinuses.

Discussion of Case 4: The glaucomatous activity was markedly greater in the right eye

* See footnote to table I.

than in the left, as indicated by tension, visual fields and the condition of the optic nerve. The sinuses on the right side were definitely more involved than were those on the left, as revealed by roentgenograms and as suggested by bacteriologic findings.

Case 5 — Glaucoma, O. U., condition in left eye more advanced than in right eye. Mr. E. L., aged 29, was first observed on March 11, 1930. He gave a history of accidental laceration of the left eyeball eight years previously, with a detached retina. No evidence of the detachment could be seen, and we question that it ever existed.

When first seen, the tension was found to be 32 mm. Hg. (Schiötz) in the right eye and 35 mm. Hg. (Schiötz) in the left eye, after instillation of homatropine and cocaine. Tension the following day, two hours after the administration of a 1 per cent solution of pilocarpine, was 19 mm. Hg. (Schiötz) in the right eye and 25 mm. Hg. (Schiötz) in the left eye. Corrected vision was 20/20-3 in the right eye, and 20/40 in the left eye. Near point of accommodation was 120 mm. for 400 mm. print in each eye. Examination of the visual fields showed a relative central scotoma for blue in each eye. The patient complained of frequent colds, nasal obstruction and chronic nasal congestion. Cultures were not taken at this time. Blood chemistry was normal except for a somewhat high uric acid value. Blood count and urinalysis were also normal.

The patient was placed on 1 per cent pilocarpine, twice a day, but this failed uniformly to control tension, especially in the left eye. In July, 1933, Dr. W. C. Bowers reported that the posterior tips of the inferior turbinates were somewhat hypertrophied, on transillumination the left antrum appeared slightly darker than the right and roentgenograms showed a slight haziness of both ethmoids. In 1936, he developed a nasal cut in the central field of the left eye as seen in figure 9 (unbroken line). In October, 1937, he suffered from labyrinthitis on the left side and was hospitalized. In December of the same year cultures from the nasal secretions yielded toxic staphylococci and those from the nasopharynx *B. coli*, toxic staphylococci and toxic streptococci. An autogenous vaccine was prepared, and vaccine was administered in minute doses at weekly intervals over a period of four years. Because the patient was allergic to ragweed, he was also given injections of ragweed extract which have been continued up to the present with beneficial results. In September, 1941, cultures from the nose and nasopharynx yielded no pathogenic organisms, after which vaccine therapy was discontinued.

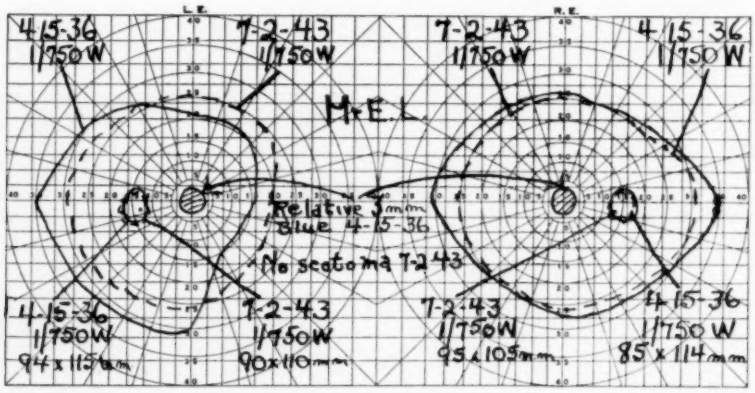

FIG. 9—Visual fields in case 5 (Mr. E. L.), chronic simple glaucoma, O. U. Unbroken line, 4/15/36, represents the visual field of the right eye taken with a 1 mm. spherical white test object at 750 mm. on a neutral gray tangent screen with 7-foot candles of artificial daylight illumination. The visual field of the left eye was also taken with a 1 mm. spherical white test object and under the same conditions. Broken lines represent the field of the right eye taken with a 1 mm. spherical white test object, showing the presence of a central relative scotoma for blue in each eye on 4/15/36 which was not found on 7/2/43.

Discussion of Case 5: Since vaccine therapy was instituted in 1937 there has been a definite improvement in tension control and at no time has it risen beyond normal limits, even though the strength of the pilocarpine has been slightly reduced (fig. 10). During this period the patient has also had nasal treatments, when indicated, and the number of colds contracted has been reduced to one or two a year. At one time there was a slight

loss of vision, but when last tested in July, 1943, corrected vision was 20/20 in each eye. Near point of accommodation has improved to 180 mm. for 300 mm. print. The visual fields have become normal as shown in figure 9 (broken line). The patient has shown marked general improvement and although 13 years have elapsed since the original diagnosis of chronic simple glaucoma was made, the visual fields are now normal, tension is well controlled by pilocarpine ¾ per cent and surgery has not been necessary.

focus of infection might affect distant organs has long since been discarded. Nevertheless, it is conceded that direct or indirect transfer of bacteria may be the modus operandi in certain cases, as for example in orbital abscess. Reber[28] states that in inflammatory diseases of the sinuses, direct infection through the bony wall may be by osteitis, periosteitis or by venous thrombosis.

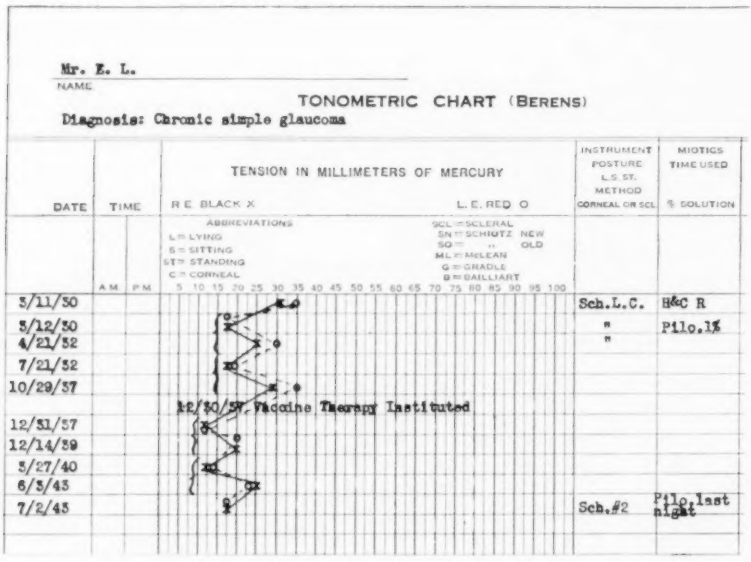

FIG. 10.—A greatly abbreviated tonometric chart of Mr. E. L. (case 5). The broken and unbroken lines indicate the tension in the left and right eyes respectively which are about equally elevated and show that the tension was stabilized better coincident with the institution of autogenous vaccine therapy.

DISCUSSION

Because we are primarily concerned with evidence supporting the proposition that sinus disease may be a causative factor in the development of chronic simple glaucoma, the mechanism by which this may be brought about does not necessarily come within the scope of this paper and it is difficult, if not impossible, to prove clinically or experimentally. However, it might be well to discuss some of the theories advanced by other investigators.

The early belief that bacterial metastasis was the only means whereby a

Anatomical changes due to infected parts have also been cited as a probable cause of certain ocular disorders. For example, infection of the sphenoid or ethmoid sinuses may affect the optic nerve because of pressure exerted on the thin separating plate of bone. Inflammation extending by direct anatomic continuity from the sinus walls to the orbit may cause pressure resulting in circulatory disturbance of nutritional vessels of the nerve. Robb[30] suggests that in contiguous inflammation from the sinuses to the eye, the ocular effect is probably due to stasis of the vascular

supply of both the sinuses and the eye. He believes "it could be explained on the fact that both the ethmoid and ciliary vessels come off the ophthalmic in immediate proximity, the ciliary just behind the ethmoid, and therefore could be readily blocked by the same process. The fineness of the anatomic structures of the eye, the ease with which small canals or spaces are blocked with exudate and with which adhesions are established, binding together minute structures of the eye, make a fertile field for great functional change associated with small pathologic findings."

Today, however, it is generally accepted that in most cases of focal infection, bacterial toxins or metabolic products are more likely to be the etiologic agents than the bacteria themselves. Limiting ourselves to sinus diseases associated with ocular disease, we are aware that there is still much dissension as to the means by which these agents, produced in the nose, may reach and affect the eye. One possibility often mentioned is by way of venous circulation, directly or indirectly. Instances in which even remote foci have been shown to affect the eye lends plausibility to indirect means of transfer. In nasal infections the venous relationship is more intimate, since, as mentioned by Reber,[28] the optic nerve and the posterior sinuses of the nose have the same source of blood supply. He points out the fact that there are two veins from the nasal mucosa which communicate by way of the angular vein with the superior ophthalmic vein and that there are arching branches across the root of the nose which connect the venous system of one orbit with that of the other. Reber[28] further states that branches from the orbicularis and lacrimal nasal duct communicate directly with orbital veins, and that these facts were ascertained by injection preparations and by radiography. He mentions the studies of Krauss and those of Birch-Hirschfield who showed that blood from the eye and orbit has a very free passage either forward or backward because of the almost complete absence of valves in the veins. He also cites Onodi who pointed out that the central vein of the retina sometimes opens into the cavernous sinus but may also pass into the pterygoid plexus.

Another channel through which products of infection may be transported is by the lymphatic system. Reber[28] feels that there is insufficient evidence to prove the presence of lymphatic spaces and lymph current in the orbital tissue. However, he cites the work of Birch-Hirschfield who demonstrated the presence of these in dogs, rabbits and apes and stated that pathological specimens gave strong presumptive evidence that such spaces exist in human orbital tissues. Benedict[3] mentions the work of Mullen and Ryder who studied experimentally the manner in which toxic material is absorbed from the sinuses. They investigated the lymph drainage of the nasal accessory sinuses and found that lymph absorption from the antra and frontal sinuses is by way of the submaxillary and internal jugular nodes. Childrey and Essex,[14] who studied the rate of absorption of toxic material, found that absorption was more rapid through severely infected mucous membranes than through normal ones. In contradiction to these opinions, Kafka[22] claims that anatomical basic facts disprove the spread of toxic material from the sinuses by lymphatics. He also throws some doubt on the theory that infection may travel by perineural sheaths since there is no direct connection between the peripheral parts and the intradural structures.

If we grant that bacterial invasion or metastasis is not necessary in order for sinusitis to affect the eye but that the toxic products of bacteria may be trans-

ferred to vulnerable points, whatever the means, there still remains a question as to how and why the eye is affected. In a number of different eye diseases, nasal pathology has been mentioned as a possible etiologic agent and yet only a small proportion of persons suffering from sinus disease have ocular involvement.

Turner[36] suggests the possibility of direct absorption of toxic products from the sinuses into the subarachnoid space, and in this way to the nerves.

Stauffer[33] has pointed out four ways in which the nerves involved in the pathology of the nose and the eye may be affected by sinus trouble. First, by direct extension of suppurative processes; second, by hyperplastic processes in the sphenoid or posterior ethmoid sinuses impinging upon the lumen of their foramina; third, by absorption of toxins directly through thin sinus walls; fourth, from the effect of reflex irritation from diseases in the nose on the vasomotor apparatus which controls circulation in the orbit, especially in the anterior pole of the eye. He pointed out that continued reflex stimulation disturbs the function and nutrition of the organ and makes it more susceptible to disease. Stauffer believes that the reason why some patients can tolerate sinus disease without ocular involvement, while others cannot, is because of variation in anatomy of the sphenoid and ethmoid cells in relation to the sensory nerves in these cavities. He has found Meckel's ganglion buried sometimes as deep as 9 mm. in the sphenomaxillary fossa, while in other cases it has been separated from the middle meatus under the posterior end of the middle turbinate only by the thickness of the nasal mucosa. He has found the same to be true of the vidian nerve. In discussing Stauffer's work, Monson stated his belief that while spectacular recoveries from ocular disease following nasal surgery or treatment might occasionally be attributed to coincidence, the percentage of favorable results are too high by the law of averages for these recoveries to be attributed to coincidence. He also suggested the possibility of an allergic factor in these cases, in which the patients become sensitized to their own bacteria or bacterial toxins. This theory is also held by Brown,[10,11] Irons[21] and Friedenwald.[20] Irons, however, presupposes an original sensitization of the eye occurring by actual bacterial embolism. Friedenwald differentiates allergy from immunity and states that sensitization has a deleterious effect upon healing because it breaks down healthy granulation tissue around the focus. We have ordinarily considered sensitization synonymous with bacterial allergy, and believe that toxins or toxic products are usually the responsible agents, rather than the bacteria themselves. Clinical studies, our experiments and those of others[4,10,11,15,26] seem to bear out this concept.

Further investigation may reveal more clearly the mechanism or mechanisms by which the eye may be affected by sinus infection or nasal pathology. In the meanwhile, as pointed out by Woods,[37] "If there is anything in the clinical picture which is suggestive of focal infection as the cause of endogenous ocular disease, operative eradication of the primary focus is indicated." In addition or in conjunction with this procedure it would seem advisable to employ immunotherapy. In some instances such therapy may render nasal or other surgery unnecessary, and in those cases in which surgery is unavoidable, desensitization may hasten recovery. This is particularly desirable in view of the fact that secondary foci may still be active, and that insofar as the sinuses are concerned, complete eradication of the infection is so often difficult to accomplish by local treatment or surgery alone.

However doubtful the relationship between ocular disease and a suspected focus of infection may be, the patient should unquestionably be given the benefit of the doubt. This should include repeated and thorough investigations for foci of infection, with local and general therapy, as well as surgery, whenever indicated.

In chronic simple glaucoma we naturally do not know whether the bacteria and their toxins play as important a role as do other factors except possibly as they may sensitize the eye and account for transient attacks of edema. However, the distribution of pigment in the eye, the postoperative tendency to mild iritis and the changes in the lens capsule might be associated with a prolonged absorption of bacterial products. It would seem as though ocular reflex vasomotor disturbances and passive congestion might be the most important factors in causing the tension to be elevated, especially during exacerbations of tension. The fact that in our series of cases elevation in tension was so often coincident with mild attacks of sinusitis on the same side seems to support this view more than the theory of invasion of the eye by bacteria or their products. It seems likely however that both factors may play a part.

SUMMARY AND CONCLUSIONS

Focal infection has been advanced as one of the possible etiologic factors in causing chronic simple glaucoma, and of the many foci suggested, infection of the nasal membranes and accessory sinuses has appealed to us as being of particular interest because of the apparent relationship of unilateral nasal pathology with unilateral glaucoma originally observed in several cases.

Of the 53 unselected cases of chronic simple glaucoma included in this study, all showed varied indications of sinus infection or nasal pathology. Thirty-seven (70 per cent) of these patients had either unilateral glaucoma or showed more signs of glaucoma in one eye than in the other. In each of these 37 patients, the nasal involvement was greater on the same side as the eye with the more active glaucoma. In six cases the glaucoma was bilateral and the sinus involvement was substantially the same on both sides. In 10 instances the signs of glaucoma were greater on one side than the other, but the nasal involvement was either bilateral or seemingly slightly worse on the side opposite that of the eye more seriously affected by glaucoma. Perhaps the most significant finding was the fact that in no instance was there marked nasal pathology on one side associated with a markedly active chronic simple glaucoma on the other side. In those cases in which there was considerable pathology, both ocular and nasal, it was either on the same side, or else the unilateral ocular condition was associated with bilateral sinus disease. Bacteriologic studies of the nasal membranes, although inconclusive, were suggestive of nasal pathology because of the high percentage of uncommon pathogens recovered.

These findings, although not conclusive, are at least highly suggestive of a close relationship between chronic sinusitis and chronic simple glaucoma. The manner in which nasal pathology or sinus infection may affect the eye has not been fully determined although vasomotor disturbance, caused by chronic hyperplastic sinusitis, is probably the more important factor. Venous stasis probably may play a part by causing mild congestion of the eye. That ocular sensitization to bacteria or their products is important is suggested by the frequent development of mild iritis following operations for glaucoma and by the finding of chronic mild uveitis in eyes diagnosed clinically as chronic simple glaucoma. Edema of the ocular tis-

tues found by Theobald and so often associated with allergic reactions may play an important role. That sinusitis can cause changes in ocular tension seems to be highly probable in view of our observation of the frequent association of attacks of ocular hypertension with exacerbations of sinusitis. In seeking the etiologic cause of chronic simple glaucoma, it is suggested that infection of the nasal accessory sinuses may be a possible factor, and that examination of the patient should include a thorough investigation of the upper respiratory tract and, if sinus disease or other causes of recurring attacks of nasal congestion and edema are found, they should be conscientiously treated.

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DISCUSSION

ALAN C. WOODS, M.D., Baltimore, Md.: Doctor Berens has presented the premise that focal infection, especially of the accessory nasal sinuses and the nasal mucosa, may be an etiological factor in chronic simple glaucoma. In an extremely well written paper, which he was courteous enough to send to me, he has very clearly presented a case for the affirmative. Great as is my personal regard for Doctor Berens and deep as is my respect for his professional and literary ability, on this one proposition propounded by him I find myself in complete disagreement. May I, therefore, present the case for the negative.

The doctrine of focal infection was brought into prominence by Billings in 1916. It was bolstered up at that time by a great deal of experimental work which has now largely been discredited. Nevertheless, it enjoyed immediately a great deal of popularity. It gave to physicians an explanation for a certain group of chronic and acute cases for which previously there had been no explanation, and it gave them something to do for patients for whom they hitherto had had nothing to offer. There followed the great era of the eradication of foci of infection: extirpation of tonsils, radical operation on the accessory nasal sinuses, extraction of teeth, both sound and diseased, removal of the pelvic organs, wild talk of gastro-intestinal infection, auto-intoxication, and then such things as colonic irrigations, and even the removal of huge stretches of the large intestine.

As a result of these operations, many patients were undoubtedly benefited, many others were unimproved and some of them were positively worse. The net result was that many patients were finally reduced to little more than the vital organs necessary for existence only to find that the original condition for which they had made the sacrifice of the various parts of their anatomies was unchanged. Gradually among internists, pathologists and bacteriologists the theory of focal infection began to fall into more or less disrepute, but among ophthalmologists it still continued to enjoy a modicum of popularity.

As the idea of bacterial metastases from the primary to the secondary focus became untenable, various other means were sought to try to identify the relationship between the primary and the secondary focus, and chief of these was bacterial toxins, which was extremely weak because very few of these organisms had exotoxin or endotoxin of any strength. Then came the more plausible explanation of bacterial sensitization. Elsewhere at considerable length, I have endeavored to present the case for and against the doctrine of focal infection, and I have ventured the rank heresy that this "Goddess of Focal Infection," whom we were raised to worship with unquestioning faith, is, after all, a very shabby creature, standing on a shaky pedestal, clad in dismal and delapidated raiment. Yet I am prepared to admit that there are a small number of cases of endogenous ocular dis-

cases where all the accumulated clinical evidence points strongly to the fact that the ocular disease is related in some manner to some focus of infection, and the most plausible explanation we have for this, which has some sound experimental backing, is bacterial sensitization.

The question before us now is, therefore, plainly, does chronic simple glaucoma belong in this group of endogenous ocular disease? Frankly, I do not believe it does. I have had a fairly wide experience in the clinical study of chronic glaucoma, and while undoubtedly many patients with chronic simple glaucoma have some indication of sinus disease and nasal infection, it is not my impression that such infection is greater among glaucoma patients than it is among any other similar age group. We must remember that, on the eastern seaboard, sinus disease and chronic nasal infection are extremely widespread, and there are very few of us in this room who are not standing convicted of nasal sinus disease if the evidence of deflected septum or the cultivation of staphylococci or a chance streptococcus from the nose is accepted as valid evidence. The normal nasal flora are alpha Streptococci, several types of gamma Streptococci, Micrococcus catarrhalis, diphtheroids, and Staphylococcus aureus and albus. A few colonies of beta Streptococci, pneumococci and H. influenzae are quite often found in the nasal flora of individuals who have no evidence whatever of the disease, either clinical or systemic, or sinus or nasal.

The mere fact that these organisms on *in vitro* resistance tests show evidence of pathogenicity, or even show evidence when injected into animals, is no criterion for believing that they are pathogenic to the host from whom they were cultured. I might recall to you the very simple experiment which is done in the first-year class in bacteriology, in which the student takes some of his own sputum and injects it into the tail of a rat, waits around for the rat to develop haemococcic septicemia and then isolates the organism. The pneumococcus is absolutely non-pathogenic to the host, and, therefore, with all due respect to Doctor Berens, and I say this regretfully, I do challenge the statement — and I quote from his paper — "that the presence of pathogenic organisms indicated nasal pathology although nasal examination and roentgen rays were negative." If I accepted this statement and routinely subjected all patients with chronic simple glaucoma to cultures of the nasal mucosa, I have no doubt our figures for nasal pathology in glaucoma patients would equal those presented by Doctor Berens. However, since only the glaucoma patients who show some clinical evidence of nasal or sinus disease or who give a positive history of such disease are routinely sent for an otolaryngological consultation, I do not find the incidence of sinus disease or nasal infection in glaucoma patients either remarkable or noteworthy.

An examination of the nasal and sinus findings reported by Doctor Berens does not disclose anything that is extremely remarkable. Undoubtedly many of these patients had actual sinus disease, but in the greater number the evidence of actual disease of the nose or sinuses is unconvincing. Such unconvincing evidence are the clinical reports of deflected septa, congestion of a turbinate, dry mucous membrane, chronic postnasal drip or "nasal mucous membranes congested at times." The report that these findings occur with increased incidence in cases of glaucoma is interesting. It may be that in an eye that already has glaucoma, edema in the posterior nasal sinuses may tend to produce a secondary rise in the tension but this is not evidence of an etiological relationship between the sinus disease and glaucoma simplex.

I think the crux of the question after all is whether or not these patients who have chronic simple glaucoma and sinus disease have their glaucoma benefited or cured by eradication of the nasal infection or by radical operation on the sinuses. In the first two cases reported by Doctor Berens there was some definite improvement in the ocular picture under treatment of the sinuses, but both of these patients subsequently required operations on their eyes. I can find no mention of the effect of treatment of the sinuses of the third and fourth cases. In the last case it was observed that the glaucoma improved and required a weaker solution of pilocarpine to control it while the patient was under vaccine therapy, but such an observation in one isolated case is not evidence.

There are two other points I might bring up. Do otolaryngologists who treat sinus disease and nasal infection from morning to night encounter chronic simple glaucoma in their patients with any undue frequency? They certainly do not in Baltimore. I have been over the question with otolaryngologists. The second question is: In Arizona and Colorado, where sinus disease and nasal infections are at their lowest ebb, is there any notable absence or decrease in the incidence of chronic simple glaucoma? I frankly do not know, but I should like to hear from ophthalmologists in Denver and Arizona on that point.

Therefore, although Doctor Berens' paper is suggestive and interesting in its effort to throw light on this obscure disease, I can find

no evidence that, to my mind at least, supports the premise that sinus and nasal infection have an etiological relationship to chronic simple glaucoma.

E. H. Wood, M.D., Auburn, N. Y.: I think that Doctor Berens is going to smile a little when I say what I have to say about this particular paper. I am one of John Evans' boys. We have found in our study of glaucoma and of sinus disease by a method known as angioscotometry that in the presence of sinus disease there is an increase in the size of the blind spot, in the size of the angioscotoma, which is very similar to the early scotomas that are found in the study of glaucoma cases. We find that with adequate treatment and drainage of sinuses the size of the scotoma decreases, and if we are successful in treating the sinus disease and draining the sinus, the scotoma returns to nearly normal size.

Likewise, in cases of early glaucoma where an increase in the ocular tension is present, it is also found that there is an increase in the size of the scotoma which, when treatment with pilocarpine is instituted, returns to nearly normal size. This similarity may or may not be something in Doctor Berens' favor, but I mention it just as another point which I think is worth stating.

W. H. Crisp, M.D., Denver, Colorado: Just as many of my patients in Colorado will say they have sinus trouble as would those in New York.

Conrad Berens, M.D., New York, N. Y.: At least I expected what I got. However, I did not expect that Doctor Woods would open himself quite as wide to my return engagement as he did. He discussed mainly focal infection and brushed aside the possible effect of unilateral congestion and edema on one eye. He also said nothing about the quiet iritis following operation for chronic simple glaucoma, or why certain of these wounds cease filtering after operation. He said nothing about the most important question of unilateral reflexes from the nasal membranes and sinuses, and little concerning our unilateral findings. He did not, as I hoped he would do, present a series of carefully studied cases; not merely his impressions from the negative side, but an impartial study. If he has accurate records with good histories of nasal pathology on which to base his statements, or if he will obtain them during the next ten years, he will find out whether the suggestion I have made has any foundation. I hope somebody will do that.

I read Doctor Woods' paper on focal infection last night, and I agree with practically everything he said in it. He does say "remove foci of infection in some cases." Doctor Woods cannot quote me as giving statistics of patients who had uveitis caused by certain known conditions, including foci of infection. I have never, as he did, put myself on record as saying that I knew that a patient had uveal disease caused by a certain condition, even sinus disease. I have considered it very problematical that one can really be certain of the etiology. Doctors Woods and Guyton, in a study of 562 cases of uveitis, said that "the patients in the study were divided into two groups, the first group consisting of 244 in whom the results of diagnostic study and the therapeutic response to treatment were so clear that a *definite* diagnosis of the etiologic factor responsible for the ocular disease appeared justified. In 31 or 12.7 per cent the uveal inflammations were due to foci of infection."

If Doctor Woods had attended my lecture on uveitis this morning, he would have heard me say that clinically I have no definite ways of determining the etiology of uveitis. The Association for Research in Ophthalmology was developed some 16 years ago because we were particularly anxious to see if we could find out anything about the etiology of uveitis. I do not have the slides with me to show the pathological reports of 437 eyes with uveitis, examined at my request at the Army Medical Museum by Colonel Ash and Mrs. Wilder, to determine how accurate our diagnoses are in chronic uveitis. Of the 437 cases, there were clinical and pathologic diagnoses of tuberculosis in 37 cases; in 30 cases there was no clinical diagnosis of tuberculosis but there was a pathologic diagnosis of tuberculosis; while in 19 cases the clinical diagnosis was tuberculosis, but there was no histologic evidence of its presence. Thirty-eight of these eyes were diagnosed clinically as being affected by chronic simple glaucoma but had definite uveitis which was really secondary. This suggests that clinically we may not always diagnose the uveal disease in chronic simple glaucoma.

Doctor Woods says that the removal of foci of infection does not cure his patients of their arthritis or uveitis, and has a very beautiful analysis of the subject in his paper. In my lecture this morning I showed a table of 162 patients with uveitis, seen in private practice, in whom I suspected that certain things might be a cause; but I made no posi-

tive assertions about the etiology of the conditions that I saw clinically. I have seldom reported complete cures of uveitis, nor have I said that I could remove all foci of infection. I believe it is difficult, if not impossible, to remove all foci of infection, and will discuss sinus disease especially. Doctor Woods does not name the bacteria found in his patients with sinus conditions, nor does he tell exactly how those sinus diseases were "cured." My experience leads me to believe that well established chronic ethmoiditis is seldom cured by any means, and that coliform bacterial infections are in some instances among the most difficult to eradicate. I have unfortunately had the opportunity of seeing men like MacKenty — I will speak of him because he is dead — and other great men engaged in the field of focal infection in sinus disease, operate on patients with chronic ethmoiditis; I have failed yet to see any of these patients, whose eyes we were observing, completely cured by surgery or postoperative treatment to the ethmoids. So much for the general problem of focal infection.

Now so far as this particular work is concerned, the point I should like to leave with you gentlemen is that in studying these records, some extending over a period of 22 years, one is struck with the frequent reports of one-sided nasal symptoms, more marked on the side of the active glaucoma. It takes a long time to go through the records, but the statistician working on them would underline certain points, for example, the frequent occurrence of inflammation or repeated attacks of trouble on one side of the nose. This association of more marked nasal disease on one side, with more active glaucoma on the same side, was found in 70 per cent of the 53 patients studied. We naturally cannot show you all those charts in detail; that would be impossible, but they are available for study. Unfortunately time permitted the presentation of only five cases here, but as was explained, these were representative of the 53 cases studied. When published, this paper will include a table containing the condensed findings in all these cases. It will be noted that in 17 instances ocular improvement was reported, usually in conjunction with successful nasal treatment. These 17 cases did not include those in which simultaneous ocular and nasal improvement was only temporary, with subsequent relapses. Particularly significant was the frequency with which periods of exacerbation or improvement of the ocular condition coincided with a similar condition in the nasal membranes or sinuses. If, as implied by Doctor Woods, eradication of a sinus or nasal infection with an associated benefit or cure of the glaucomatous condition would be suggestive of a relationship between the two diseases, would not exacerbation of the nasal infection associated with marked ocular disturbance be equally suggestive?

The discussion seems to narrow down to this: We have made the simple suggestion that from a very careful study of the records, our patients seem to have an association between attacks of sinus trouble on one side and attacks of ocular hypertension on the same side. In suggesting infection of the nasal membranes or acessory sinuses as a possible etiologic factor in chronic simple glaucoma, we did not intend to imply that it might be the only etiologic factor. There are probably other necessary conditions involved, to which the addition of an infected sinus may be the inciting factor. We suggest merely that in the presence of predisposing conditions, whether congenital or acquired, sinusitis may bear the same relationship to chronic simple glaucoma as, for example, emotional strain does to attacks of acute glaucoma. In certain cases it is even possible that such an infection may be the prime etiologic factor, if one is to attach any importance to the work of Dr. F. Stauffer who pointed out the anatomical variations in location of the sensory nerves in relation to the sphenoid and ethmoid cells, and the consequent variations of ocular reaction that might, therefore, be anticipated when these sinuses are infected. This might also be one answer to Doctor Woods' question as to why do not all who suffer from sinusitis become also affected with glaucoma or other intra-ocular diseases. We do not see how we could use controlled cases in this study.

Doctor Woods also suggested that in the event of a relationship between sinusitis and endogenous ocular disease, one might expect the incidence of glaucoma to be lower in those regions in which sinus disease and nasal infections are at their lowest ebb. In 1941, Dr. Daniel Snydacker reported on the geographical incidence of glaucoma throughout the world and stated that in the United States it appears "that the incidence of glaucoma is higher in the urban centers of the East and Northeast, and diminishes almost in direct ratio to the population in progressing westward into the mountain area, to increase again in the populous area of the Pacific Coast." What Doctor Snydacker seemed to attribute to closely populated areas might just as easily be attributed to climatic conditions, especially when one recalls that even in the middle west there are large crowded cities. Climatic conditions affecting the incidence of glaucoma

are apparently the same as those affecting sinusitis. A number of investigators have reported the proportionally higher incidence of glaucomatous attacks during the late fall and winter months, while others have shown that marked changes in barometric pressure are frequently associated with attacks of acute glaucoma. That these same periods and climatic conditions are also associated with an increase in sinus and nasal exacerbations is too well known to question.

Doctor Woods questions another point — the significance of streptococci in the nose and quotes the group from Cornell with whom I worked in studying arthritis and chronic infection. We have made careful studies of the bacteriology of the nasal membranes on both sides for a number of years. We have come to the conclusion, from what we find later when the roentgenograms show trouble, and when the rhinologists are able to make a diagnosis, that the finding of streptococci in the anterior nares is suggestive of pathology. The same thing apparently applies to the coliform bacteria. We have not found that coliform bacteria are normally present in the upper respiratory tract, although the men with whom we worked, who were leaders in the field of focal infection, said that we would find coliform bacteria anywhere in normal people. So far as we are concerned, we do not find this to be true, and the work of others who have taken the trouble to make bacteriological studies and statistical reports of their findings supports our opinion. Several physicians came to me this morning and said that their patients had made the diagnosis of their nasal difficulty from their own symptoms and insisted on sinus treatment. Later apparently the patients were proved to be correct in this respect.

Doctor Woods appears to discount bacterial toxins as being a significant factor in focal infection, because "very few of these organisms have endotoxin or exotoxin of any strength." Toxic staphylococci and coliform bacteria, organisms often found in nasal infection, both produce a powerful exotoxin. Exotoxic streptococci, such as the Lancefield Group A Hemolytic streptococcus, would be expected to produce acute rather than chronic disease. The fact that the patient is able to tolerate the toxins of his own bacteria for a long time before showing symptoms of sensitivity may demonstrate the comparatively low pathogenicity of the organisms, but does not indicate that the toxins are harmless, especially after sensitization has been produced.

For the comfort that Dr. E. H. Wood has given me, I will assume that possibly what he had to say is more than suggestive, because we have noted these things ourselves. In reference to one of these patients, I had hoped that somebody would speak about that particular type of field, because although he did not have a greatly enlarged blind spot, he had a condition that I thought was more likely to be found in sinus disease than glaucoma. He did have baring of his blind spot, but he had no contraction of his nasal field. We have often found patients with enlarged blind spots and little, if any, contraction of the nasal field although ocular hypertension had been present for some time. In patients with field changes of this type associated with ocular hypertension, we make especially careful study of the upper respiratory tract.

In conclusion, I have for years thought there was some association between sinus disease and chronic simple glaucoma and maybe some day somebody will prove it.

EPIDEMIC KERATOCONJUNCTIVITIS

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An epidemic of acute conjunctivitis has occurred in the United States during the past few years. Since it first appeared in epidemic proportion in the shipyards on the Pacific coast it came to be called "shipyard conjunctivitis" or "California conjunctivitis." It appears that the disease was present in California before it became epidemic, since Hobson[8] in 1938 found 16 cases of superficial keratitis in a veterans' hospital at San Fernando, California. Rieke[15] estimated that 600 cases occurred in one shipyard in Oregon in 1941, and de Roetth[4] at the same time called the disease "Epidemic Keratoconjunctivitis" and called attention to its association with superficial punctate keratitis. Hogan and Crawford[9] summarized the disease in a report of 125 cases from the San Francisco bay region. It is interesting to note that although the disease was widespread on the Pacific coast, in relatively few cases did corneal opacities develop.

Fuchs[5] in 1889 emphasized the corneal changes. From the epidemic that occurred in Vienna, he reported 38 cases of a disease he called "Superficial Punctate Keratitis." The keratitis was preceded by acute conjunctivitis, and the disease he described is similar to our present disease. In his description he stated that it was a form of superficial keratitis related to herpes febrilis corneae but not associated with the formation of vesicles. These corneal changes had a predilection for the superficial layers and were concentrated in the central portions of the cornea. He also emphasized that the disease was frequently unilateral and that following recovery from the acute conjunctivitis pain, photophobia and lacrimation were common. Adler,[1] Van Reuss[18] and others described a similar disease during the same period.

The next reported cases came from Bombay, India, where in 1900 Herbert[7] observed 26 cases. American physicians visiting India during the period between 1900 and 1920 have spoken of seeing patients with severe conjunctivitis associated with corneal opacities that was not trachoma. In 1920 Kirkpatrick[12] made a short report of the disease while Wright[20] and later Kirwin[13] described extensive epidemics in Madras, India. The disease became widespread in India and China between 1920 and 1933. Outbreaks of a similar nature occurred in India, China and Malaya from 1933 to 1936, in Germany from 1938 to 1940, and in Malaya again in 1935 to 1938. Hamilton,[6] in a survey of eye disease in Tasmania, described numerous cases of a similar disease. Holmes[10] in 1941 estimated 10,000 cases in Oahu, Hawaii.

From all reports it seems probable that the present epidemic began in Malaya, spread to Hawaii, then to our West coast.

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During the early part of this study convalescent serum was drawn and prepared under the supervision of Doctors Perkins and Korns of the New York State Department of Health. The major portion of the convalescent plasma was prepared by the blood bank of the Presbyterian Hospital under the supervision of Dr. John Scudder to whom I am deeply indebted. (The method of preparation of plasma has been repeated elsewhere.) Dr. Murray Sanders and Mrs. Rose Alexander tested all specimens, donors and recipients for the presence of antibodies.

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EPIDEMIOLOGY OF CASES IN NEW YORK

Perkins, Korns and Westphal[14] have summarized the epidemiology of the outbreak of epidemic keratoconjunctivitis in New York state as occurring in Schenectady. They reported the findings in several thousand cases. The acute conjunctivitis in their series was bilateral in 56 per cent of the cases. Corneal opacities developed in 85.5 per cent of the cases and in 28.4 per cent of these the condition was bilateral. In 92 per cent of the cases corneal opacities developed between the seventh and the 23rd day of the disease. In the examination for corneal opacities a simple spot light was used in most cases. The incidence of opacities may have been greater if the corneal microscope had been used. Even though the slit lamp was not used, the incidence of corneal opacities was much higher than in any of the previously reported groups.

In their series the highest incidence of infection occurred in the medical dispensary. Although it could not be stated definitely that the dispensary was the primary source of the individual infection, it seemed suggestive.

Doctors Sanders, Gulliver, et al.,[16] also suggested that some of the patients may have contracted the disease after visiting a physician's office.

I am convinced that a number of individuals have undoubtedly contracted the disease in various clinics. Several case histories are definite and give an accurate period of incubation. In the control cases where definite exposure was known, this incubation period was usually five or six days. Two cases are of interest. A physician was called upon one evening to remove an ocular foreign body. He found a moderate amount of edema of the upper lid with some chemosis and conjunctival injection. After a careful search he was unable to find a foreign body. Six days later the physician developed a similar condition which became a typical case of epidemic keratoconjunctivitis. Subsequent examination of the patient showed the presence of many corneal opacities. The second case was that of an attending ophthalmologist at Vanderbilt Clinic. He was demonstrating cases of conjunctivitis to a group of medical students. One case was diagnosed as early epidemic keratoconjunctivitis, but the diagnosis was somewhat obscure. Five days after the demonstration, he noted a foreign body sensation with considerable discomfort in his own left eye. The symptoms gradually became worse accompanied by considerable chemosis and edema of the upper lid. The disease followed the usual course, and corneal opacities developed on the eighth day.

Two other incidents have been called to my attention. The first involved the use of the tonometer and the second the use of contact lenses. The diagnosis of early epidemic keratoconjunctivitis is frequently presumptive so that intraocular tension was taken in these cases in order to rule out a diagnosis of acute glaucoma. The tonometer was then used on two other patients with chronic glaucoma, both of whom developed epidemic keratoconjunctivitis five and six days afterwards. Possible contagion from the fitting of contact lenses has been called to my attention by Dr. F. H. Adler of Philadelphia. He states that two patients who were given trial fittings of contact lenses developed epidemic keratoconjunctivitis on the fourth and sixth day following these fittings.

Although clinics and dispensaries are being incriminated as sources of transmission of epidemic keratoconjunctivitis, many more cases have been seen which had not had a previous contact either with a physician's office or a clinic. In order to determine the source of infection, a large number of patients have been questioned carefully. Most of these have admitted a mild upper res-

piratory infection associated with the disease. The common occurrence of upper respiratory infection may indicate that this association is merely incidental. However, in crowded places, it is not unlikely that eye infections as a result of contamination from nasal droplets may occur. With this in mind two patients are of interest.

Miss N. was an operating room nurse in a large local hospital. She came in contact with very few people. On her day off she went on a shopping tour and visited several large department stores. To her knowledge she did not encounter anyone with acute conjunctivitis. One week after her tour, acute conjunctivitis developed in her left eye which became typical epidemic keratoconjunctivitis.

Miss C. P. was a director of Occupational Therapy in a large hospital for chronic diseases, and her directing duties were such that she rarely came in contact with anyone except the personnel of her department. She took a train trip and likewise did not knowingly come in contact with any cases of acute conjunctivitis, but eight days later developed an acute phase of what subsequently proved to be epidemic keratoconjunctivitis. These cases illustrate the possibility of infection either by fomites or droplets from individuals who may or may not have the disease.

Early in the study the occurrence of foreign bodies and foreign body sensation in the eyes of patients developing the disease was considered of importance. Because of the frequency with which cases of corneal foreign body occur, it has not been possible to determine accurately the number of cases of epidemic keratoconjunctivitis which have developed as a result of corneal foreign bodies. In comparatively few of these cases did epidemic keratoconjunctivitis develop. It is doubtful that the foreign body carried the infection and much more likely that the patient was inoculated with the disease when the foreign body was removed. This was brought to my attention in reviewing histories of a number of patients. One patient is of interest. Mr. P., a hospital employee, got something in his left eye on his way to work. He reported to Vanderbilt Clinic, and under pontocaine anesthesia a small foreign body was removed from the cornea. Sulfathiazole ointment was placed in the eye, and the eye was covered. The following morning the corneal abrasion was healed, and the eye looked normal. Six days later he returned with an acute conjunctivitis, and a tentative diagnosis of epidemic keratoconjunctivitis was made. This was subsequently confirmed when typical corneal changes occurred.

In summarizing the epidemiology, I believe that ophthalmologists and physicians in general should be meticulously careful of the cleanliness of their hands and their instruments. It has been reported to me by Dr. Murray Sanders that his virus survives in most of the common solutions used in ophthalmology. The virus, however, deteriorates quite rapidly and loses considerable virulence when it is at room temperature for three to six hours.

Most of the common antiseptics have proven to be of little value. Detergents are superior to any of the other antiseptics. Droplet and fomite infections cannot be controlled.

ETIOLOGY

Sanders[17] isolated a filtrable virus from scrapings of the conjunctiva of two patients with acute epidemic keratoconjunctivitis. He found that the virus could be isolated by inoculating conjunctival scrapings into tissue culture and then later injecting the tissue culture intracerebrally into mice. To date his work has not been confirmed. This part of the study was an attempt to isolate a virus and confirm the work of Sanders.

METHOD OF STUDY

Scrapings from the conjunctiva of patients with the disease were suspended in fluid tissue culture consisting of serum ultrafiltrate, balanced salt solution and embryo mouse brain tissue. These cultures remained dormant. Mouse brain maintains viability but shows little evidence of growth. The inoculated cultures were allowed to stand at room temperature for periods varying from five to 10 days. The tissue and fluid were then placed in the tissue culture grinder and carefully ground until no evidence of tissue remained. Six white Swiss mice (Rockland strain) were each inoculated with ground tissue culture, .05 cc. intracerebrally and .15 cc. intraperitoneally, and ½ cc. was placed in a fresh tissue culture. The mice were examined daily for evidence of encephalitis. Twenty-five attempts were made to isolate a virus. Several cultures were contaminated with pathogenic bacteria and were discarded. Other attempts did not give rise to any symptoms in mice. The brains of sick mice were ground, and four or five healthy mice were inoculated with tissue culture. Attempts were made to step up the potency of virus to a concentration where it would consistently produce death in mice. My virus did not develop such potency. Table I illustrates the trend of the disease in my mice. Mice recovering from inoculation with my virus were given intracerebral inoculations of Sanders' virus and showed high recovery rate as compared to the control (see Table II).

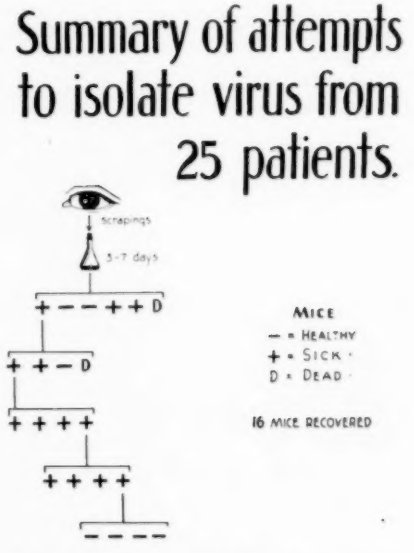

Table 1

TABLE II.

Recovered Animals Inoculated with Sanders Virus.

Control series

	10^{-3}	10^{-4}	10^{-5}	10^{-6}
1.	----	----	----	----
2.	D+++	+++D	--++	----
3.	DDD	DDD	++DD	++--
4.			DD	DD++
5.				DD
6.				

Recovered series

10^{-3}	10^{-4}	10^{-5}	10^{-6}
----	----	----	----
----	----	----	----
++--	D+--	----	----
DD--	D--	----	----
D-	--	----	----
-	--	----	----

TABLE IIa.

Results of Intracerebral Inoculation of Epidemic Keratoconjunctivitis Virus into Forty Recovered Mice.

First test:	10^{-3}	10^{-4}	10^{-5}	10^{-6}
6/24	*****	*****	*****	*****
6/25	*****	*****	*****	*****
6/28	dddd*	ddd**	*****	*****
7/3	*	**	*****	*****

Second test:	10^{-3}	10^{-4}	10^{-5}	10^{-6}
6/29	*****	*****	*****	*****
6/30	*****	*****	*****	*****
7/2	d****	*****	*****	*****
7/3	dddd	*****	*****	*****
7/5		dd***	*****	*****
7/6		d**	*****	*****
7/13		**	*****	*****

Control:	10^{-3}	10^{-4}	10^{-5}	10^{-6}
6/29	*****	*****	*****	*****
6/30	*****	*****	*****	*****
7/2	ddd**	*****	*****	*****
7/3	dd	*****	*****	*****
7/5		ddddd	dd***	*****
7/6			ddd	d****
7/7				dddd

RESULTS

There is little reason to doubt that I obtained an agent from patients suffering from epidemic keratoconjunctivitis. This agent produced transient symptoms of encephalitis in mice and could be passed by serial passage from mouse to mouse and to tissue culture and back to mice. Its potency was not increased by serial passage. Since it produced a sub-lethal infection it might, however, produce immunity. To test this hypothesis, 40 recovered mice were given to Doctor Sanders. He states that "40 mice which survived intracerebral injection were re-injected with 10^{-3}, 10^{-4}, 10^{-5} and 10^{-6} dilutions of stock epidemic keratoconjunctivitis virus. A similar number of normal mice of the same age were injected as controls. The results, Table IIa, were as follows: In 40 normal mice death occurred in all

previously mentioned dilutions. In the test animals deaths occurred only in groups which received dilutions of 10^{-3} and 10^{-4}. No symptoms and no deaths were observed in those animals which were injected intracerebrally with dilutions 10^{-5} and 10^{-6}. To me this strongly suggests that you have been dealing with a virus similar to one isolated in this laboratory."

I believe that although I was unable to develop a lethal virus, my agent was a virus similar to that of Doctor Sanders. It seems possible that my agent may be a high dilution of a virus similar to that of Sanders.

THE CLINICAL DISEASE

The symptomatology and findings present in epidemic keratoconjunctivitis are those of hyperacute conjunctivitis. The disease may be divided into three stages. Stage I is an acute phase in which the primary symptoms are those of foreign body sensation with moderate itching and burning. The most striking features are edema of the upper lid, chemosis and edema of the semilunar fold and caruncle. The foreign body sensation described during this acute phase may be so marked and come on so suddenly that the individual is sure he has a foreign body in his eye. Edema is almost always quite striking, especially of the bulbar conjunctiva. In some instances the chemosis is so marked that the bulbar conjunctiva is exposed even when the eye is closed. The majority of the cases show moderate chemosis. The chemosis is associated with hyperemia of the conjunctival blood vessels. During this stage the eye frequently feels enlarged, and many individuals have complained of pain and discomfort on ocular rotation. This may indicate changes in Tenon's capsule.

The disease enters the second stage in approximately 48 hours. This is characterized by development of large follicles in the conjunctiva and considerable subconjunctival infiltration, particularly in the lower lid, but the upper retrotarsal fold and fornices are also involved. The follicles in the conjunctiva are large, translucent and tend to be oval in shape. Between follicles, there is present in the conjunctiva moderate papillary hypertrophy with considerable hyperemia. During this phase there is nearly always a palpable, slightly tender pre-auricular lymph node. There may also be submental and submaxillary lymphadenopathy. It is during this phase that upper respiratory symptoms are present associated with a scratchy sensation in the throat. In some of the cases there has been evidence of follicular pharyngitis. During both the acute and follicular stages of the disease, tearing is a prominent symptom and not infrequently the tears are blood stained. After five to six days the edema decreases, and the lymphadenopathy may or may not disappear. At times the lymphadenopathy will have disappeared by the sixth day while again it may persist for several weeks. The tenderness of the lymph nodes rarely lasts beyond the sixth day. During this phase of the disease a pseudomembrane may develop on the conjunctiva of either the upper or lower lid.

Frequently, small hemorrhages are seen on both the bulbar and palpebral conjunctivae, but these are not so severe as those seen in acute pneumococcal conjunctivitis. In the more severe cases many times the pseudomembrane develops on the lower palpebral conjunctival surface. This pseudomembrane is thin when compared to the thick pseudomembrane of streptococcal conjunctivitis, but much thicker than the very fine pseudomembrane associated with allergic conjunctivitis. A membrane may also be present on the upper lid. A pseudomembrane was present on the third to the fifth day in 67 per cent of the

cases. The pseudomembrane, when examined microscopically, was composed almost entirely of epithelial cells with mononuclear cells scattered throughout. When the pseudomembrane is light yellow in color it contains many more mononuclear cells and degenerating epithelial cells. This was usually present on the fifth to the seventh day. In its early development the slightly opaque translucent membrane is composed almost entirely of viable superficial conjunctival cells. I was unable to demonstrate virus bodies (inclusion bodies) in any appreciable number although when stained by the Giemsa method an occasional blue staining body could be seen in the cytoplasm. Victoria blue stain failed to demonstrate any virus.

The third stage of the disease is characterized by the continuing follicular conjunctivitis associated with beginning corneal changes. These corneal changes produce symptoms of photophobia and blurring of vision. At times the blurring may be quite marked before any corneal opacities appear, and occasionally during the beginning of this phase there may be a mild superficial punctate keratitis. Superficial points on the cornea have been rare. Loss of vision may be due to slight edema of the corneal stroma. I have noted particularly with the narrow beam of the slit lamp a slight difference in the thickness of two corneae before corneal opacities develop which may account for the early loss of vision. Corneal opacities usually begin to form on the seventh or eighth day of the disease. There is considerable variation in the manner in which the corneal changes occur. The most common types begin as minute, discreet, subepithelial dots. These dots increase in number forming opacities approximately one millimeter in diameter. These grey opacities are located just beneath the epithelium and seem to lie in or on Bowman's membrane. They are usually circumscribed and circular and have a sharp border. The epithelium over the surface is occasionally slightly raised but does not stain with fluorescein (1 per cent).

The opacities are concentrated in the central portion of the cornea, usually most numerous over the pupillary area. Occasionally the opacities are so numerous that they coalesce to form a grey opacity in the superficial cornea. In spite of their numbers and their close proximity it is almost always possible with the bio-microscope to see that they are composed of discreet nebula. When they are numerous one can see that not all opacities lie in the same level of the cornea — some being on Bowman's membrane and some under it. Photophobia is usually marked, and there is considerable loss of vision when the opacities are numerous. The corneal opacities usually increase in number and size from the eighth to the 21st day, during which time the conjunctival findings are decreasing. The marked hyperemia gradually decreases, and the eye is white after three or four weeks. The blurring of vision and photophobia, especially in artificial light, continues for several months, and many individuals complained that if they did excessive near work the bulbar conjunctival vessels became hyperemic and there was a return of mild symptoms.

Several patients have been followed for periods up to 18 months. The clearing of the opacities is a gradual process. The fine dots composing the opacities decrease in number, and the density gradually decreases. As the density decreases the visual acuity is increased. Vision usually becomes normal but "fuzzy" long before the opacities have disappeared. The number of opacities decrease without leaving any perceptible scar. In nearly all the patients followed for a long period one or two opacities have remained visible with the slit lamp.

In none of the patients has any permanent loss of vision resulted.

PATHOLOGY

The pathology has been studied by examining conjunctival scrapings. No conjunctival biopsies have been made. The conjunctival epithelial cells appear normal and show no evidence of definite inclusion bodies, although in a few cases blue staining bodies have been found in the cytoplasm of the cells. Bodies similar to these have been found in other types of conjunctivitis so their significance is unknown at the present time. The most striking findings have already been pointed out by Hogan and Crawford[9] in their earlier reports. The findings in my cases have been similar. Large and small lymphocytes with a few large mononuclears predominate in the scrapings. Polymorphonuclear leukocytes are rarely found and then only when there was a bacterial contamination. From the findings in scrapings one would suspect that the follicle contributes many of the lymphocytes but also that the entire conjunctiva was densely infiltrated with them. One striking finding in my cases has been the unusual number of broken inflammatory cells. This has led to a possible point of differentiation of the disease from Beal's conjunctivitis in which lymphocytes also predominate but in which fragile cells are rare. The pathology in mice has been discussed by Sanders.[17] In mice the Sanders virus gives rise to a mild lymphocytic encephalitis.

BACTERIOLOGY

Cultures have for the most part been sterile. In some cases xerosis brucelli and other diphtheroids have been recovered. In several cases a non-pathogenic Staphylococcus albus has grown, while in a few cases Staphylococcus aureus was found. These latter cases may show a few polymorphonuclear leukocytes in scrapings. Early in the disease cultures were invariably sterile while later, after considerable local medication, Staphylococci were almost always found.

TREATMENT

Every effort has been made to control carefully the cases receiving treatment. Early in the epidemic I concluded that it was not possible to make an absolute diagnosis of epidemic keratoconjunctivitis before corneal opacities had developed. The value of treatment had to be based on rapidity of recovery and the prevention of corneal opacities. If these criteria were used, the original diagnosis would certainly be open to question unless better diagnostic methods were available. Fortunately, Sanders[17] showed that patients recovering from the disease possessed a substance in their blood sera which when mixed with dilutions of his virus prevented death in mice, which was not the case when normal sera treated in a like manner was used. This virus neutralizing substance could be expressed by the reverse of the dilution factor. If the patients' serum-virus mixture diluted 10^{-2}, 10^{-3}, 10^{-4}, 10^{-5}, and 10^{-6} killed mice in a dilution of 10^{-2} and 10^{-3} without producing symptoms in higher dilutions, and the normal serum-virus mixture diluted likewise killed all mice through dilution up to and including 10^{-6}, then the patients' sera had 10,000 units of neutralizing substance. It was found by Sanders[17] that the patients with other types of conjunctivitis did not have these substances in their sera. Patients with Beal's conjunctivitis were especially interesting in that they did not show antibodies to Sanders' virus in their sera. The finding of antibodies by Sanders suggested the use of convalescent serum or plasma.

In our control series of treated patients two questions arose. Did the in-

travenous convalescent plasma stimulate the antibody formation? What effect would normal plasma have on the disease? In order to test the effect of convalescent plasma on antibody formation, five patients were given 50 cc. intravenously and one patient 100 cc. (Table III). Blood was taken from these patients at intervals up to three months and tested for the

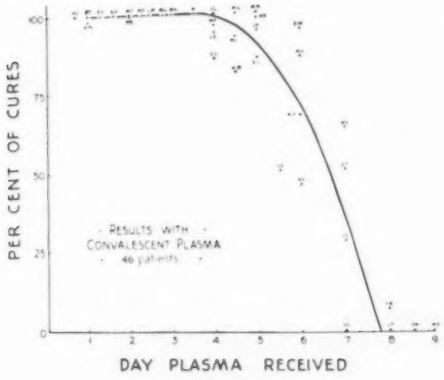

Fig. 1

presence of antibodies. In only one patient was there any evidence of neutralizing substances and this one received 100 cc. of plasma. She was a physician with a severe pneumococcal conjunctivitis in her only eye. She was given this amount of plasma since she had not responded rapidly to local sulfonamide therapy. She has not been available for a final check on the antibody titer of her serum, but she had very little antibody at the end of two months. The conclusion made from the tests was that 50 cc. of convalescent plasma was not sufficient to effect the test for antibodies.

Convalescent plasma has been given to 47 patients in whom a diagnosis of epidemic keratoconjunctivitis was made. Samples of serum were obtained from these patients at intervals (Table IV). In many of the cases serum was obtained during the acute phase of the disease, and, although the list is not complete, none had neutralizing antibodies to the virus. The neutralizing substances became apparent in from one to three months. Case 19, Miss H. S., is of particular interest because she did not show any response to the plasma, and she was slow in developing antibodies. Even though she had many corneal opacities she did not show any signs of antibodies until three months after her corneal opacities had developed. When the results are expressed in a graph it is seen that little or no effect can be expected if

TABLE III.

Results of Intravenous Convalescent Plasma in Patients with Other Types of Conjunctivitis.

Case	Name	Diagnosis	Amount of conv. E. K. Plasma in C.C.	Resultant Titer of Serum
1.	C. A.	Staph. conjunctivitis	50	Neg.
2.	J. C.	Pneumo. "	100	10+?
3.	P. H.	Burns of skin	50	Neg.
4.	G. D.	Acute conjunctivitis of unknown etiology	50	Neg.
5.	G. T.	Erythema multiforme	50	Neg.
6.	L. P.	Chronic Staph. Keratoconjunctivitis	50	Neg.

TABLE IV.

Results of Convalescent Plasma Therapy.

Case	Name	Days of Disease Before Conv. Plasma	Amount of Conv. Plasma	Results of Corneal Change	Final Titer of Patient's Serum
1.	G. T.	8	50	1+	10,000
2.	M. T.	3	40	Neg.	10,000
3.	B. T.	2	20	Neg.	10,000
4.	M. N.	5	40	Neg.	10,000
5.	A. S.	6	30	1+	10,000
6.	J. S.	6	40	3+	10,000
7.	D. S.	4	15	1+	1,000
8.	J. L.	6	25	Neg.	10,000
9.	H. T.	5	50	Neg.	1,000
10.	M. F.	4	40	Neg.	1,000
11.	E. L.	5	50	Neg.	1,000
12.	W. K.	1	50	Neg.	1,000
13.	B. M.	2	50	Neg.	1,000
14.	L. M.	9	50	4+	10,000
15.	H. H.	1	50	Neg.	1,000
16.	G. S.	3	50	Neg.	1,000
17.	W. M.	4	50	1+	1,000
18.	E. Y.	1	50	Neg.	1,000
19.	H. S.	7 and 9	100	4+	100
20.	A. H.	4	50	Neg.	1,000
21.	M. G.	2	50	Neg.	1,000
22.	M. P.	2	50	Neg.	10,000
23.	M. L.	3	50	Neg.	1,000
24.	L. P.	9	50	4+	10,000
25.	J. F.	5	50	Neg.	1,000
26.	M. G.	4	50	Neg.	100

the plasma is given after the seventh or eighth day (Fig. 1). Good results can be expected if the plasma is given before the fifth day. After the fifth day the results are unpredictable, some will be good while others will be poor.

In order to have a series of controls, five patients with epidemic keratocon-

TABLE IV.—Continued.

Case	Name	Days of Disease Before Conv. Plasma	Amount of Conv. Plasma	Results of Corneal Change	Final Titer of Patient's Serum
27.	W. G.	2	50	Neg.	1,000
28.	H. C.	3	50	Neg.	100
29.	S. S.	5	50	3+	1,000
30.	H. B.	5	50	1+	1,000
31.	W. S.	7 and 9	100	4+	10,000
32.	L. N.	4	50	Neg.	1,000
33.	W. L.	3	50	Neg.	1,000
34.	W. F.	9	50	4+	100
35.	W. T.	3	50	Neg.	1,000
36.	P. P.	2	50	Neg.	1,000
37.	T. G.	3	50	Neg.	100
38.	S. S.	4	50	Neg.	1,000
39.	L. B.	5	50	Neg.	1,000
40.	D. D.	4	50	Neg.	100
41.	B. B.	3	50	Neg.	Lost
42.	P. H.	2	15	Neg.	100
43.	F. H.	2	20	Neg.	1,000
44.	K. M.	3	30	Neg.	100
45.	M. K.	4	20	Neg.	100
46.	P. G.	3	15	Neg.	100
47.	G. W.	6	20	1+	100

Degree of Corneal Opacities:
1 plus equals less than 3 opacities.
2 plus equals 3 to 5 opacities.
3 plus equals 5 to 10 opacities.
4 plus equals 10 or more opacities.
(The first 10 of these patients were previously reported—(17).

junctivitis were chosen and given 50 cc. of normal plasma intravenously (Table V). The normal plasma had no effect on the progress of the disease nor on the development of serum antibodies.

The majority of patients were treated symptomatically. However, 35 were treated with sulfathiazole, 25 with a five per cent concentration of sulfathiazole ointment and 10 with a three per cent solution of sodium sulfathiazole sesquihydrate. Clinically none of these patients were benefited by the therapy, and many of them complained that the local use of sulfathiazole ointment increased their symptoms. In a therapeutic test

with sulfathiazole and sulfadiazine, white mice were fed sulfathiazole by mouth for two days and then were injected with various dilutions of the virus. (Table VI.) Eight mice were injected with each dilution so that 48 mice in all were used. Sulfathiazole, 0.5 grams, and sodium bicarbonate, 1.5 grams, were mixed with 100 grams of casein food, and each mouse received five grams of the mixture a day. This is a standard method of protecting mice against streptococcal and pneumococcal infections. There was no evidence of protection against the virus. Forty-eight mice were then treated in a similar manner with sulfadiazine (Table VII). There was likewise no evidence of protection against the virus. From the clinical evidence and from experimental evidence, sulfathiazole and sulfadiazine had no effect on the virus.

TABLE V.

Results of Normal Plasma on Epidemic Keratoconjunctivitis.

Case	Name	Day of Disease	Amount of Normal Plasma	Resultant Corneal Changes	Final Serum Titer
1.	L. P.	4	50	4+	1,000
2.	M. B.	6	50	4+	10,000
3.	B. E.	3	50	4+	1,000
4.	A. S.	5	50	4+	1,000
5.	L. R.	2	50	4+	100

TABLE VI.

Sulfathiazole Feeding.

5/17 Mice fed .5 gram of Sulfathiazole and 1.5 grams of Sodium bicarbonate in 100 grams of Casein food. Each mouse had 5 grams of the mixture a day.

5/19 Injected	10^{-1}	10^{-2}	10^{-3}	10^{-4}	10^{-5}	10^{-6}
5/20	**** ****	**** ****	**** ****	**** ****	**** ****	**** ****
5/22	ddd* ****	**** ****	**** ****	**** ****	**** ****	**** ****
5/23	d dddd	dddd dddd	dddd dddd	dddd ddd*	**** ****	**** ****
5/24				d	ddd* ****	d*** ****
5/25					d d***	*** ****
5/26					dd*	*** ****
5/27					d	*** ****

Local penicillin ointment (500 units per gram) was used in three patients with no effect. Huang[11] has reported to me that penicillin has no effect on the virus when tested *in vitro*.

A 48 and 20 mg. per cent solution of tyrothricin has been used in 10 patients. It was difficult to evaluate the treatment since nearly all patients experienced some symptomatic improvement. The symptomatic improvement, however, was about the same as that reported by patients using pontocaine and adrenalin. The course of the acute phase of the disease seemed to be shortened, although all 10 patients developed a large number of corneal opacities. Since there was some indication that tyrothricin might be beneficial in the treatment of the disease, several *in vitro* tests were attempted. The entire series of experiments is reported in Table VIII. The results are difficult to evaluate since it would appear that the tyrothricin-virus mixtures in higher dilutions when allowed to stand at room temperature did not kill mice. Few deaths occurred as a result of injecting mice with the dilutions of 10^{-5} and 10^{-6} when the dilutions had been allowed to stand at room temperature two hours. When these results are compared with the control series there seems to be a deterioration of the virus when it is allowed to stand at room temperature. There are, however, other factors involved since deaths in mice occurred erratically. From the findings in mice one must conclude that there is little or no effect of the tyrothricin on the virus. In the clinical cases even though the acute conjunctivitis was decreased in severity the disease ran a normal course, in spite of the fact that two of the patients were given tyrothricin on the first day of the disease.

A total of 296 patients with epidemic keratoconjunctivitis have been treated. Of these, 209 were treated symptomatically. In all but two of these patients corneal opacities developed. In the two

TABLE VII.

Sulfadiazine Feeding.

5/17 Mice fed .5 gram of Sulfadiazine and 1.5 grams of Sodium bicarbonate in 100 grams of Casein food. Each mouse had 5 grams of the mixture a day.

5/19 Injected	10^{-1}	10^{-2}	10^{-3}	10^{-4}	10^{-5}	10^{-6}
5/20	**** ****	**** ****	**** ****	**** ****	**** ****	**** ****
5/22	dd** ****	**** ****	**** ****	**** ****	**** ****	**** ****
5/23	dd dddd	dddd dddd	dddd dd**	ddd* ****	**** ****	**** ****
5/24			dd	d dddd	dd** ****	**** ****
5/25					dd ****	**** ****
5/26					dd**	d*** ****
5/27					dd	d** ****

TABLE VIII.

Action of Tyrothricin Against the Virus EK

15 cc of Z solution to 1.5 cc of Tyrothricin.
0.1 cc of this mixture to each dilution.

I. Injected immediately on mixing the drug and the virus.

5/13 Injected	–3	–4	–5	–6
5/14	o***	****	d***	****
5/15	dd*	dd**	***	****
5/17	d	**	***	****
5/18		d*	ddd	****
5/19		d		****
5/25				****

II. Drug and virus mixed and allowed to stand at room temperature for two hours.

5/13 Injected	–3	–4	–5	–6
5/14	dd**	****	****	d***
5/15	d*	d***	****	***
5/17	*	***	d***	***
5/18	*	d**	***	***
5/19	*	**	***	***
5/20	d	d*	***	***
5/25		*	***	***

III. Drug and virus mixed and allowed to stand at room temperature for three hours.

5/13 Injected	–3	–4	–5	–6
5/14	****	****	****	****
5/15	ddd*	****	****	d***
5/17	*	d***	d***	***
5/18	d	***	***	***
5/25		***	***	***

TABLE VIII.—Continued.

18.5 cc of distilled water to 1.5 cc Tyrothricin.

5/13 Injected				
5/14	d*****			
5/25	*****			

22.5 cc distilled water to 2.5 cc Tyrothricin.

0.1 cc of this mixture to each dilution.

I. Injected immediately on mixing the drug and the virus.

5/13 Injected	–3	–4	–5	–6
5/14	****	****	d***	o***
5/15	d***	****	***	***
5/17	dd*	****	***	***
5/18	d	ddd*	***	***
5/19		d	d**	***
5/25			**	***

II. Drug and virus mixed and allowed to stand at room temperature for two hours.

5/13 Injected	–3	–4	–5	–6
5/14	d***	****	****	****
5/15	***	****	****	****
5/17	***	****	****	****
5/18	dd*	o***	****	****
5/19	d	o**	****	****
5/21		d*	****	****
5/25		*	****	****

III. Drug and virus mixed and allowed to stand at room temperature for three hours.

5/13 Injected	–3	–4	–5	–6
5/14	****	****	****	****
5/18	ddd*	****	****	****
5/19	d	****	****	****
5/25		****	****	****

TABLE VIII.—Continued.

13.5 cc of distilled water to 1.5 cc Tyrothricin.
0.1 cc to each dilution.

I. Injected immediately on mixing the drug and the virus.

5/20 Injected	–2	–3	–4	–5	–6
5/22	****	****	****	****	****
5/24	dddd	ddd*	d***	****	****
5/25		d	ddd	ddd*	dd**
5/26				d	**
5/27					d*
5/28					d

II. Drug and virus mixed and allowed to stand at room temperature for two hours.

5/20 Injected	–2	–3	–4	–5	–6
5/21	****	****	dd**	o***	****
5/22	****	d***	**	***	d***
5/23	****	***	d*	d**	***
5/24	ddo*	***	*	**	***
5/25	d	dd*	*	**	d**
5/26		d	*	**	**
5/31			*	**	**

III. Drug and virus mixed and allowed to stand at room temperature for three hours.

5/20 Injected	–2	–3	–4	–5	–6
5/21	****	****	****	****	****
5/24	ddd*	****	****	****	****
5/25	d	dddo	ddd*	****	****
5/26			d	****	****
5/31				****	****

TABLE VIII.—Continued.

Virus alone without Tyrothricin.

I. Injected immediately.

5/20 Injected	–2	–3	–4	–5	–6
5/21	****	****	****	****	****
5/23	oo**	d***	****	****	****
5/24	dd	ddd	ddd*	ddd*	dd**
5/25			d	d	d*
5/26					d

II. Virus allowed to stand at room temperature for two hours before injection.

5/20 Injected	–2	–3	–4	–5	–6
5/21	****	****	****	****	****
5/24	ddo*	d***	****	****	****
5/25	*	ddd	d***	****	****
5/27	d		od*	d***	****
5/28			d	dd**	****
5/31				*	****

III. Virus allowed to stand at room temperature for three hours before injection.

5/20 Injected	–2	–3	–4	–5	–6
5/21	****	****	****	****	****
5/24	ddo*	do**	****	****	****
5/25	*	dd	do**	****	****
5/27	*		d*	****	****
5/28	*		d	****	****
5/31	*			****	****

patients in whom corneal opacities did not develop there was considerable doubt as to the accuracy of the original diagnosis, and, unfortunately, neither of these patients has been available so that their blood could be examined for antibody titer. Since the original diagnosis was made in their cases during the height of the epidemic, and although the diagnosis is doubtful, these two cases are still classed as epidemic keratoconjunctivitis. Corneal opacities, then, developed in 99 per cent of the patients who were not treated with convalescent plasma, while in those treated with convalescent plasma corneal opacities developed in approximately 30 per cent. As has been shown before, in the majority of the patients first treated after the fifth day of the disease corneal opacities developed. The failure of convalescent plasma to effect cures after the disease is established is borne out by the experience of others in the treatment of other virus diseases. With other virus diseases, convalescent plasma, or serum, is of value if given during the incubation period, or may be of benefit if given during the first days of the illness.[3] This experience seems to be indicated by my figures.

COMMENT

The disease, epidemic keratoconjunctivitis, while new to us, has occurred in epidemics elsewhere in the world. In the previous epidemics the corneal opacities were stressed. It is difficult to make a diagnosis before the corneal opacities develop. As in trachoma, where a pannus must be present before a diagnosis of trachoma can be made, I believe that subepithelial corneal infiltration characteristic of epidemic keratoconjunctivitis must be present before an absolute diagnosis is possible. Since the patients develop immune bodies to the virus in a sufficient titer to be measured, this is another means of diagnosis. The antibody titer can be detected only long after the disease has run its course and is, therefore, of no value in making an early diagnosis. The diagnosis during the acute phase of the disease, then, is presumptuous and must remain so until corneal opacities develop or virus neutralizing substances can be measured.

CONCLUSIONS

1. Diseases similar to epidemic keratoconjunctivitis have been reported at intervals since 1889.
2. Our present epidemic probably began in Malaya in 1938 from whence it spread to Hawaii and the continental United States.
3. The clinical disease may be divided into three stages:
   a. Abrupt onset of edema associated with a foreign body sensation.
   b. Follicular conjunctivitis and pseudomembrane formation with lymphadenopathy and upper respiratory symptoms.
   c. Retrogression of conjunctivitis with the development of corneal opacities.
4. The etiology is probably a filtrable virus and my findings substantiate those of Sanders.
5. Patients recovering from the disease have immune bodies to the virus in their serum.
6. Convalescent plasma, or serum, if given before the fifth day of the disease, is the treatment of choice.
7. Tyrothricin probably has no effect on the virus.
8. The sulfonamides and penicillin have no effect on the virus.
9. Corneal opacities developed in 99 per cent of the patients treated symptomatically, but in only 30 per cent of cases treated with convalescent plasma.
10. Epidemiology is obscure but certainly in a few epidemics the physi-

cian was responsible for the transmission of the disease. Simple hygienic measures consisting chiefly of cleanliness of the hands seem to prevent transmission.

DISCUSSION

HARRY S. GRADLE, M.D., Chicago, Illinois: There is little that I can add or detract from Doctor Braley's complete presentation, and consequently I shall confine myself to questions in the hope that he may have the answers by this time next year.

While the majority of papers concerning epidemic keratoconjunctivitis deal mostly with the conjunctival aspect of the disease, the corneal phase is of even greater interest and of the utmost importance. Why is there the characteristic corneal involvement in some cases and not in others, and why does the percentage of such involvement vary with the locality? Are the corneal lesions direct virus infections and if so what is the channel of admission? Why should the corneal infiltrates assume a characteristic appearance and maintain that appearance throughout their existence?

Just how these questions are to be answered, I do not know. As far as I do know, there has been only one report concerning microscopic examination of the corneal infiltrate. This was by Rosemarie Günther of the Löhlein Clinic, published in the 103rd volume of the Klinische Monatsblaetter für Augenheilkunde in 1939. This eye was blind and was enucleated because of painful iridocyclitis. The blindness had developed subsequent to retinal detachment. There was no clinical evidence of epidemic keratoconjunctivitis except for "in the center of the otherwise normal cornea are some round infiltrates with serrated edges, lying sub-epithelially." The picture was complicated by marked conjunctival and ciliary injection with vessels well advanced over the limbus onto the cornea. Microscopic examination revealed collections of lymphocytes, histiocytes and fibroblasts lying between Bowman's membrane and the corneal epithelium in the areas that corresponded to the infiltrates visible biomicroscopically. Such collections of cells lifted the epithelium from the basal membrane into small humps, in the larger of which the epithelium was so stretched that only a thin layer remained as a cover. Toward the basal membrane was a homogeneous, structureless mass which the author considered to be bits of broken-down Bowman's membrane. Only where such breaks occurred did the infiltrate involve the superficial corneal layers.

In the discussion, Günther mentioned that clinically the corneal lesions stained with fluorescein in a stippled manner, a finding that is not present in the reports concerning cases in this country. Several other clinical facts were at variance with the findings reported here so that one is led to question whether or not this really was a case of epidemic keratoconjunctivitis. Microscopic findings in cases of various types of keratitis punctata superficialis resembling those of epidemic keratoconjunctivitis have been reported by several authors. Wehrli in the Klinische Monatsblaetter f. Augenheilkunde in 1906 described such cellular infiltrates, all of which were anterior to Bowman's membrane, while in Verhoeff's case (Archives of Ophthalmology, 1912) the infiltrates had penetrated the basal membrane and resembled necrotic masses in the anterior parenchyma. So, until more evidence is at hand, we must regard the microscopic picture of the corneal lesions of epidemic keratoconjunctivitis as being unconfirmed.

EDMOND L. COOPER, M.D., Detroit, Michigan: Doctor Braley is to be complimented upon his work on epidemic keratoconjunctivitis. His special interest in all conjunctival diseases led him early to a study of the disease, and he alone, among all investigators, has come forward with something really constructive in the matter of treatment of the disease. As he mentioned, except for treatment consisting of injections with convalescent plasma, all other forms of treatment seem to be useless. However, this plasma is available only when large epidemics exist, because it can hardly be expected that the plasma will be available in sufficient quantity where the epidemic is slight or in isolated cases.

It is my opinion that large epidemics can be prevented, and to support this, I should like to relate our experience with the disease in Detroit. In September, 1942, it became apparent that keratoconjunctivitis was occurring in industrial centers located closer and closer to Detroit. Accordingly, with the cooperation of the Detroit Department of Health, the Medical Department of Chrysler Corporation established a program for handling eye cases in an attempt to prevent the spread of the disease should it appear. Subsequently, a modification of this program was adopted by nearly every industry in Detroit. We feel that as a direct result of this program, the disease was kept at a minimum.

In the modified program, these points were included:

1. Form letters containing information regarding the clinical characteristics of the disease, its mode of spread and various recommendations regarding treatment were sent to ophthalmologists, industrial physicians, first aid personnel and safety personnel in industry, industrial nurses and industrialists.

2. It became evident according to reports in the literature and from conferences which were held with New York industrial physicians who had encountered a large outbreak of the disease that meticulous care and cleanliness are essential in the control of the disease, and that person-to-person contact in the ophthalmic department is the most frequent cause for the disease to spread. Therefore, a clinic for diagnostic and treatment purposes was established by the Detroit Department of Health to which industry was invited and had the privilege of sending suspicious cases. The real purpose of this clinic was to keep patients with epidemic keratoconjunctivitis out of the ophthalmic sections of industrial medical departments.

3. A strict technic for treatment of communicable diseases was established in the ophthalmic section of every industrial medical department. Nurses assigned to treat communicable diseases were furnished by the Board of Health to assist. I think this point is probably the most important one.

4. Patients with the disease were strictly excluded from work until all acute signs of the disease had disappeared.

5. Patients with the disease were informed fully regarding its communicability and regarding the precautions to be taken.

In Detroit the first case of epidemic keratoconjunctivitis was detected early in December, 1942. Upon investigation, 16 other cases were diagnosed in a small industrial plant employing 120 persons. This proportion was a great deal higher than that in any other industrial plant in the Detroit area. These 16 cases of epidemic keratoconjunctivitis occurred before the medical personnel of this small organization was aware that it was dealing with a communicable disease and before our control measures were instituted.

The Board of Health established a clinic for diagnosis and treatment of the disease on December 19, 1942. During the first six months 1,096 patients were examined, and a diagnosis was made of keratoconjunctivitis in 247. The largest concentration of cases in any industrial plant after our control plan had been established was 60 cases in a plant employing 90,000 persons. In another plant employing 35,000 persons, 29 cases were diagnosed, and in one employing 42,000 persons, 20 cases were diagnosed.

When these percentages are compared with those of other localities in which the disease became widespread, it is evident that the control procedures employed in the Detroit area played an important role in reducing the incidence of the disease there.

As for drugs and methods of treatment, success was not attained with the use of any except tyrothricin, which was administered in the last 99 cases. Tyrothricin seemed to shorten the course of the disease and to lower the incidence of corneal opacities. However, consideration should be given to the fact that tyrothricin was administered when the epidemic was diminishing rapidly and when the virulence of the organism perhaps was becoming less.

Convalescent plasma was not used.

It is my opinion, then, that the best treatment for epidemic keratoconjunctivitis is prophylaxis, and this can be accomplished by the establishment of a program such as I have outlined.

ARTHUR J. BEDELL, M.D., Albany, N. Y.: The impression has gone abroad that this is an epidemic that has spread from the west coast to the east coast. When the situation is analyzed it is found that in Honolulu one physician reported 10,000 cases and another, Dr. F. J. Pinkerton, whose report is not in press, saw 4,000 or 5,000. The outbreaks were in Portland, in the San Francisco Bay area and the Los Angeles district. The next epidemic was in Schenectady, New York. There were practically no cases along the Atlantic coast, although there are many large shipyards in that section, nor were there any in the shipyards located on the Gulf of Mexico. In fact, with the exception of the report just made from Detroit, there has been no other great outbreak in the United States. The epidemic has not gone from coast to coast.

In Schenectady there are two large industrial plants, and I am informed that the percentage of infected employees was the same in each and, further, that the same percentage of the general population contracted the disease. This is a fact that has not been given sufficient publicity.

It was a pleasure to hear Doctor Braley's discussion on epidemic keratoconjunctivitis. However, how can one make a diagnosis of keratoconjunctivitis when corneal involvement is not present? It is a fact that several conjunctival diseases resemble the one under discussion but in those diseases the cornea remains clear. Why then are some cases diagnosed keratoconjunctivitis even before the expiration of the incubation period which is, as Doctor Braley stated, four, five, six or seven

days? I have seen some cases develop 14 days after exposure.

The epidemic seems to be over. I think Doctor Braley will agree to this. We now see very few cases, and yet new employees are coming into these same shops under the identical conditions which formerly existed.

A. E. MAUMENEE, M.D., Baltimore, Md.: I should also like to congratulate Doctor Braley on his excellent report concerning epidemic keratoconjunctivitis. In order to stress the importance of some laboratory diagnostic procedure, such as the neutralizing antibody test, or the isolation of the E.K.C. virus itself, I should like to describe a condition that is indistinguishable from epidemic keratoconjunctivitis in many of its stages.

Herpetic conjunctivitis is mentioned but not described in detail in any of the standard French, German or English ophthalmological textbooks. I do not believe that the condition is so rare as this would seem to indicate, however, for during the past year at the Wilmer Institute, the herpetic virus has been isolated from six cases of conjunctivitis and keratoconjunctivitis.

Herpetic conjunctivitis usually begins with the feeling of a foreign body, in the eye, just as in epidemic keratoconjunctivitis. Within a few days, there is marked edema and redness of the conjunctiva. Within three or four days, follicles occur in the palpebral conjunctiva with an occasional pseudomembrane. The conjunctivitis may then clear without any corneal involvement, or the disease may be more protracted with development of subepithelial corneal infiltrates. Scrapings of the conjunctivae of these patients show a predominance of mononuclear cells, and the bacterial cultures are sterile.

The patient may also have systemic manifestations such as those present in epidemic keratoconjunctivitis. The corneal infiltrates which develop in cases of herpetic conjunctivitis differ somewhat in the earlier stages from those of epidemic keratoconjunctivitis in that they stain superficially with fluorescein. Not infrequently, small dendritic papules extend from these infiltrates. After a period of two to three weeks, the conjunctivitis clears and the remaining corneal infiltrates are indistinguishable from those of epidemic keratoconjunctivitis. I, therefore, agree with Doctor Gradle in that there are no pathognomonic corneal scars for epidemic keratoconjunctivitis.

(Slide.) This is a drawing made from a patient from whom I isolated the herpes virus, and you can see that the infiltrates are practically identical with the late scars of epidemic keratoconjunctivitis.

(Slide.) The next slide shows that the position of these infiltrates is just under the epithelium in Bowman's membrane.

There are a few points that will aid in the differentiation of herpes from epidemic keratoconjunctivitis. If the lid margins of the patients with herpes are examined with the slit lamp early in the day, small vesicles may occasionally be noted on the lid margins. If the cornea is stained daily with fluorescein, a superficial staining of the infiltrates may be noted. If a dendritic pattern extends from these round infiltrates, the diagnosis is easy. There is also a slight loss of corneal sensation in the herpetic corneas and, in the six cases that I have seen, all have been unilateral. Of the eight cases of herpetic conjunctivitis described in the literature, all have been unilateral.

By far, the best method of making a differential diagnosis is to inoculate a rabbit's cornea with conjunctival scrapings. The virus of epidemic keratoconjunctivitis does not take on a rabbit's cornea whereas the herpetic virus takes in almost a hundred per cent of the cases.

ALSON E. BRALEY, M.D., New York, N. Y.: I should like to thank the discussers for their kind remarks.

Doctor Gradle has asked some embarrassing questions. I am not certain whether the infiltrates in the corneae of persons affected with this disease are absolutely characteristic. In epidemic keratoconjunctivitis the infiltrates are predominately in the center of the cornea, near the pupillary area, whereas in filaria, a disease not mentioned, similar infiltrates occur near the margin of the cornea. Nothing is known concerning the histologic appearance. I do not know whether the lesions actually contain the virus, but I suspect that the virus is present in the corneal epithelium, and that the opacities seen in the cornea with the slit lamp, which are, by the way, composed of very fine dots, may represent infiltration of lymphocytes into the cornea.

No one has been able to isolate the virus from the corneal epithelium.

Nearly all viruses are obligatory intracellular parasites and therefore are fixed in the cell. I believe that the virus undoubtedly enters the cell at approximately the third to the fourth day of the disease, and after the virus is once in the cell, treatment with convalescent plasma is of no value.

I believe that the shape of the opacity in the cornea is dependent primarily upon collections of lymphocytes which are present in the

epithelium or in Bowman's membrane. This remains to be proven.

Doctor Cooper's discussion is excellent. He has carried out a very important piece of work in Detroit in preventing the spread of the disease where it could be spread if it were given an opportunity.

As to the method by which the disease has spread from the west coast to the east coast (if that is true, and I rather doubt it), it so happens that a great many workmen were sent out from many of our industrial plants to Hawaii shortly after the whole affair started, and many of these men returned not only to our west coast but to our east coast. Possibly the epidemic started simultaneously throughout the United States.

As far as the results of therapy are concerned, I am unable to confirm a diagnosis of epidemic keratoconjunctivitis when corneal opacities are not present.

However, I believe that the neutralizing antibodies to the virus that are present in the plasma certainly indicate the probable presence of the disease, and since a large number of those samples confirm the diagnosis, we feel that the test has some diagnostic value. All the first 13 patients receiving the plasma before the fifth day of the disease eventually developed positive neutralizing antibodies in their sera. I feel that that is at least some evidence that they probably had the disease. Whether our convalescent plasma was really of any value or not, I do not know. In 30 per cent of the patients to whom the plasma was administered corneal opacities developed, but they were given the plasma after the fifth day of the disease.

Doctor Maumenee's discussion is rather remote. The differentiation between herpes and this disease is to me not of any particular importance, because I have not seen this type of superficial keratitis associated with herpes. It must be always borne in mind that the herpes virus is present not only in the eye but in the mouth in a great many patients, and it is possible to isolate a herpes virus from the mouths of many of you in this room by simply taking a small amount of material and inoculating a rabbit's cornea or injecting it into the cerebellum of mice.

There is one pleasant thought that I should like to leave with you. The corneal opacities eventually clear up and rarely is there any prolonged disturbance of vision. It may take 18 months or two years; but eventually they will disappear.

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THE ESOPHAGEAL MANIFESTATIONS OF PELLAGRA

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THE DAY has long since passed when pellagra was thought of only as a disease characterized by the classic "three D's"—dermatitis, diarrhea and dementia. As stated by one of our country's outstanding investigators,[4] "classic pellagra is a syndrome affecting the skin, alimentary canal and the central nervous system. It is a disease attacking all strata of society, all races and both sexes." It is further stated by Spies[3] that to the naked eye the resulting changes appear to be only skin deep, and perhaps no single factor has so retarded the comprehension of this syndrome as the overemphasis placed upon the skin lesions. It is now agreed that pellagra is a systemic disease or general disease affecting all the cells of the body.

The first authentic reference to this interesting disease was made by Don Gaspar Casal, physician to Philip V of Spain, in 1725. His classical description of the skin lesions and the digestive and nervous manifestations was the first reference to pellagra that appeared in medical literature. He called the disease "mal de la rosa" or the "sickness of the rose" and thought it to be atypical leprosy. It was next recognized and reported in 1771 by Frapolli, an Italian. Following these original reports many cases throughout Europe were reported, where this dread disease reached epidemic proportions. Seventy-five thousand cases in Rumania alone were said to be present in 1912. In 1864, Gray[1] of Utica, New York, reported the first case of pellagra in the United States. In 1907, Dr. George Searcy[2] gave a marvelous description of the alimentary tract symptoms, skin lesions and the nervous and mental manifestations of pellagra, and his dissertation will always remain as one of the classics of medical literature.

The medical literature is now filled with multiple reports of a vast amount of research carried out on the etiology, signs, symptoms and treatment of this interesting disease. However, in a careful study of the literature I have been unable to find any description of the mucosal lesions in the esophagus of persons suffering from pellagra. During the last two years I have had 17 pellagrins referred for esophagoscopic examinations, all of whom were complaining of severe dysphagia. These patients were from 24 to 72 years of age. All have been residents of Alabama for a number of years. The large percentage of them have made their home in the rural districts, and their diet has consisted largely of salt pork, black eyed peas, corn bread, biscuit, and syrup.

Their history reveals that anorexia, burning sensation of the tongue and abdominal pains are the usual original complaints. This is followed by intense glossitis, stomatitis, gingivitis and enteritis which gradually result in marked dysphagia. The discomfort in swallowing became so intense in this group of persons that 12 could not take any solid food whatsoever when first seen in consultation and seven persons experienced extreme discomfort whenever they swallowed liquid.

Examination of these patients revealed characteristic glossitis in every instance. Many presented a roughened,

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erythematous, desquamating dermatitis, bilaterally symmetrical, appearing chiefly on the dorsum of the hands, on the elbows and on the neck. Several presented "hemangioma like" lesions of the conjunctivae.

In each case under local anesthesia a 9 mm. esophagoscope was passed. All presented an esophageal mucosa which was intensely hyperemic and in some cases edematous. Multiple tiny ulcerations of the esophageal mucosa were noted in nine cases. A barium meal was given to eight patients during which fluoroscopic examination revealed many small constricted areas along the course of the esophagus. These constrictions occurred in areas where ulcerations were found on esophagoscopic examination. A tumor or foreign body was not found in any case.

TREATMENT

Each patient was given 150 mgm. of nicotinamide, 15 mgm. of thiamin chloride and 15 mgm. of riboflavin daily for three months. Subsequently, they have been given a normal daily maintenance dose of vitamins considered essential for health.

RESULTS

Following this treatment the patients in this group could swallow solid food without experiencing dysphagia in from three weeks to two months. Each one's general health improved tremendously. There has been constant improvement in the dermatitis and stomatitis in all cases.

Five of the cases will be reported. The other 12 are so similar in all respects that reports of them would merely mean repetition.

Case 1. — B.E.C., female, white, aged 60. This patient has been a life-long resident of rural Alabama. She complained of severe dysphagia for a period of six weeks. There was no history of hematemesis or dark colored stools. She had constant substernal pain, and her weight had decreased 20 pounds during the preceding eight weeks. Examination revealed roughened erythematous lesions on the dorsum of each hand and wrist. The tongue was hyperemic, and its borders were smooth and glistening. A 9 mm. esophagoscope was passed, and the esophageal mucosa was found to be severely hyperemic throughout its entirety. Numerous small punctate ulcerations were found on the surface of the mucosa. Evidence of neoplasm was not noted. She was given the routine treatment, and her dysphagia disappeared in four weeks. She is now eating a perfectly normal diet without discomfort, and she has gained 16 pounds in weight.

Case 2. — W.M.B., female, white, aged 69. This patient had experienced dysphagia constantly for three months. The distress had become so severe that she had been unable to swallow any solid food during the two weeks prior to examination. Superficial examination revealed bilateral symmetrical roughening of the dorsum of each wrist, a hyperemic tongue with smooth borders and dryness and hyperemia of the pharynx. A 9 mm. esophagoscope was passed. The esophageal mucosa was found to be severely hyperemic. Edema was noted at the edge of three small ulcerations found on the surface of the mucosa. The routine treatment was prescribed, and the patient was also given 100 mgm. of thiamin chloride intravenously at weekly intervals by her home physician. The dysphagia gradually subsided, and she was able to swallow without discomfort two months after treatment was begun.

Case 3. — L. L., female, colored, aged 27. When this patient was first seen she had been unable to swallow anything except clear fluids for one week. She had experienced difficulty in swallowing since childhood when an esophageal stricture as the result of swallowing lye

accidentally had occurred. However, she had noticed increasing dysphagia for three months prior to examination. Because of her distress on swallowing as a result of the esophageal stricture, the patient had partaken of only semi-solid foods for several years. A 6 mm. esophagoscope was passed, and three separate constricted areas were found in the esophagus. The esophagoscope was passed by these areas with difficulty. The mucosa was markedly hyperemic, and small ulcerations were found just above each constriction. She was given the routine treatment, and dilatations of the esophagus were carried out twice weekly with the Edwin N. Broyles type of esophageal dilator. Following the fourth dilatation she was able to swallow solid food, and her dysphagia had diminished considerably. A No. 28 steel Broyles dilator now can be passed with ease, and re-examination with the esophagoscope reveals the esophageal mucosa to be normal in color and without ulceration. This treatment has been carried out over a period of three months.

Case 4. — T.B.H., male, white, aged 72. This patient was first seen May 7, 1943, with a history of dysphagia of five months' duration, substernal pain, burning sensation of the tongue and a weight loss of 20 pounds. The patient was given a barium meal, and fluoroscopic examination revealed the esophagus to be dilated except for a constriction in the mid-portion and another at the lower end. A 9 mm. esophagoscope was passed. The esophageal mucosa was severely hyperemic, and ulcerations were found in the mucosa at the midpoint and at the lower end, which corresponded to the site of the constrictions seen on fluoroscopic examination. Evidence of neoplasm was not found at any point in the esophagus. The routine treatment was prescribed, and by August 8, 1943, the patient was having no dysphagia whatsoever.

Case 5. — M. McK., female, white, aged 68. This patient was first seen August 11, 1940, with a complaint of a burning sensation of the tongue, substernal pain and fullness, marked weight loss, bizarre pain in the extremities and diarrhea. Examination revealed an emaciated white female with symmetrical dermatitis of the dorsum of each wrist, smooth, beefy-red tongue, gingivitis and a hyperemic, dry pharynx. Small hemangioma-like dilatations of the blood vessels were present on the conjunctivae. A 9 mm. esophagoscope was passed. The esophageal mucosa was inflamed, and numerous small ulcerations were noted along the entire length of the esophageal mucosa. She was given the routine treatment for four months, following which she has been on a daily maintenance dose of vitamins. Her dysphagia has gradually disappeared, and when the patient was last seen, July 9, 1943, she appeared to be a different person. The dermatitis had disappeared, she had no dysphagia and had gained approximately 40 pounds in weight.

CONCLUSIONS

1. Pellagra, although a reportedly ancient disease, is present in a large number of patients seen today. It is a disease which affects all tissues of the body and occurs in young and old persons of either sex.
2. A diet insufficient in nicotinic acid, thiamin chloride and riboflavin seems to be its chief etiological factor.
3. This disease frequently causes severe hyperemia and ulceration of the esophageal mucosa which produce intense dysphagia.
4. The inclusion of large amounts of nicotinamide, thiamin chloride and riboflavin in the diet of persons suffering from the disease will produce cessation of the symptoms and restore the sufferer to health.

I would like to express my appreciation to Dr. Tom Spies and Dr. James S. McLester for their advice in the treatment of these patients. The colored slides are shown through the courtesy of Dr. J. W. McQueen.

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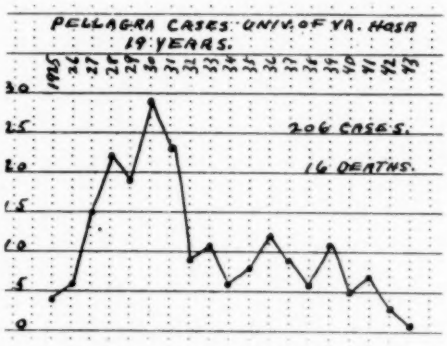
DISCUSSION

FLETCHER D. WOODWARD, M.D., Charlottesville, Va.: In reviewing the case histories of patients with pellagra admitted during the past 19 years to the University of Virginia Hospital at Charlottesville, Virginia, it was noted that the majority were from the rural areas, of the poorer class and had subsisted on an inadequate diet. However, chronic alcoholism was the underlying factor in several instances.

Many of these patients, of course, complained of dysphagia. However, due to the fact that they were referred to the Medical Service and that most physicians are not especially esophagus minded, this complaint was attributed largely to the lesions of the tongue and mouth. In only two instances was the esophagus examined fluoroscopically, and in both of these the examination revelaed a normal esophagus. Since most of the esophageal lesions are no doubt minor manifestations when compared to the picture as a whole, I do not feel critical of the Medical Service for not exhibiting more zeal in studying the esophagus in these cases.

During the past 19 years 206 cases of pellagra have been seen at the University Hospital; 16 patients died in the hospital, and autopsies were done on eight. Esophageal lesions were not noted except in one case, in which there were many submucosal hemorrhages. A more careful examination would no doubt have shown many superficial ulcerations.

It was interesting to note the large number of cases of pellagra admitted during the depression years, in contrast to those admitted during the more prosperous years. This interesting observation is shown graphically.



A graph showing the marked increase of pellagra during the depression years.

Judging from this small series of cases, the State of Virginia seems to be either relatively pellagra-free or our doctors are overlooking the diagnosis in many instances. However, patients affected by the subclinical and milder forms are not hospitalized, which no doubt accounts for the small number.

I wish to thank Doctor Fisher for calling our attention to the frequency with which esophageal lesions occur in patients affected with pellagra, and I feel sure that in the future we will see that more fluoroscopic and esophagoscopic examinations are done, in order better to understand this phase of the disease.

FRANCIS E. LEJEUNE, M.D., New Orleans, La.: I am frank to confess that I have never examined the esophagus of a pellagrin and therefore feel very incompetent to enter into this discussion.

Doctor Fisher comes from a community in which pellagra is fairly common, and he has availed himself of the opportunity presented by carrying out a study of the esophagus in a series of cases and accurately determining the fact that with certain given symptoms multiple ulcerations are usually present in the mucosa of the esophagus.

In attempting to prepare a discussion of this paper I reviewed Dr. Seale Harris' "Clinical Pellagra" and was surprised to find only two short references to the esophagus.

In the first reference Doctor Harris states that in cases of early pellagra there is a dryness of the esophagus. The other reference consists of a terse statement that examination of the esophagus reveals evidence of chronic inflammatory changes, sometimes with secondary ulcerations. Doctor Fisher has definitely proved the existence of esophageal ulcerations in those patients complaining of certain symptoms. This observation is an important one and adds much to our further knowledge of pellagra. However, one questions the advisability of esophagoscopy when the mucosa of the esophagus is markedly hyperemic, intensely dry and in some cases edematous as stated by Doctor Fisher. The general accepted treatment for patients affected with pellagra is with nicotinic acid, riboflavin and thiamin chloride plus a proper diet. This therapy will control and cure lesions of the esophagus, according to Doctor Fisher's experience. The existence of certain symptoms such as glossitis, stomatitis, gingivitis and enteritis is usually followed by dysphagia. When these symptoms occur, it should be presupposed that lesions or ulcers have occurred in the esophagus, and in view of the fact that no therapeutic advantage is offered by an examination of an already pathological esophagus, the general accepted therapy for pellagra should be instituted without the necessity of esophagoscopy.

HEARING AIDS

WALTER HUGHSON, M.D.
ABINGTON, PENNSYLVANIA

AT THE close of World War I there were an estimated 40,000 aural casualties, of all degrees of hearing impairment, returned to civilian life after discharge or resignation from the armed forces of the United States. At the present moment there are six times as many men in the American Armed Forces than there were in the last war, and these are scattered throughout the world. On a purely numerical basis we may expect a quarter million aural casualties in this war. But there are many other factors to be considered. The hazards to hearing have increased to an immeasurable extent. It can properly be assumed that no combat aviator will return to civil life at the war's end with normal hearing, despite the fact that his induction into this branch of the service required perfect hearing. Aural infection and complications of contagion are undoubtedly under better control, but acoustic trauma, the effects of high altitude flying and its corollary, acute pressure changes related to dive bombing and fighting, and finally the remote effects of the administration of serum and antitoxin and prophylactic drug therapy will increase the ratio of aural casualties to a truly appalling extent. In the last war the percentage of aural casualties was two per cent of the total. This same numerical ratio would give the quarter million estimated in this war. That the actual number will be much greater can hardly be questioned.

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Otological Research Laboratory, Abington, Pa.

The Army and Navy Surgeon General's Offices realize the importance of the problem, and have taken steps to deal with the situation adequately. The Veterans Bureau is preparing for this influx of soldiers who will eventually be returned to civilian life with a handicapping hearing loss. Quite properly the various organizations whose primary purpose is caring for hard of hearing and deafened civilians have taken steps to deal with this serious post-war problem. Leagues for the Hard of Hearing and established rehabilitation bureaus for handicapped people will play an essential role in its final solution.

However, the entire situation is primarily a medical one and more specifically an otologic one. For this reason the American Academy of Ophthalmology and Otolaryngology has appointed a committee whose purpose shall be to insure every possible aid for these aural war casualties. This great group of men will be returned to their home communities with a serious handicap, namely a hearing loss. They will have been cared for in an expert manner before discharge from the Armed Forces. But this care cannot terminate at that point. On their return to civilian life an entirely new problem presents itself and an entirely new adjustment must be made.

It is rather an unhappy commentary on our concern for the deaf that a war must be the means of stirring us into decisive action. But unquestionably much good will result from this final effort provided we as otologists come to realize our responsibility to the deaf and the hard of hearing.

Any disability, whether incurred in civilian life or as a result of military service, requires effort toward rehabilitation. The American Academy of Ophthalmology and Otolaryngology has as one of its appointed committees one which has to do with the Prevention and Amelioration of Deafness and as a subcommittee one concerned with the Rehabilitation of the Deaf. According to Webster an individual can be rehabilitated by "being restored to a former capacity," in this case "hearing," or as a second definition "to fit to make one's livelihood again." This discussion will be concerned primarily with rehabilitation.

The medical treatment of deafness is, we all know, relatively unimportant and ineffective. Surgical treatment, if applied according to accepted precepts, is from a numerical point of view insignificant. It is unlikely that any acquired war deafness will be amenable to surgery of any type other than that employed for the correction of chronic infection.

Civilians require rehabilitation now so that they may play their part in the general war effort. The aural casualties of this war are already being returned home as incapable of further military duty. The immediate handling of both types of hearing loss is identical. There is not time for medical treatment of uncertain value or promise. For the civilian, elective surgery under war condition is an improper hazard to undergo.

There is but one ready solution to this present and impending problem, and that is the proper fitting of an adequate hearing aid. The Council of Physical Therapy of the American Medical Association through its Committee of Consultants approves or withholds approval of commercial hearing aids. The otologist is ethically bound to endorse only such approved aids.

Before proceeding further it may be well to modify the foregoing statements. The present situation is one of emergency which may last a matter of months or years. Every scientifically minded otologist and every research otologist has before him the hope that new and immediately effective therapeutic technics may be developed. Were this not so our specialty would be a completely stagnant one. But for the immediate control of the present emergency it is felt that this exposition of therapeutic control of deafness is the only feasible one.

War casualties and civilian deaf require immediate help now. As otologists we have more or less disregarded the particular professional obligation of intelligently advising our patients in the selection of an appropriate and efficient hearing aid. The reasons for this indifference are obvious. The fitting of hearing aids is time consuming, infinite patience is requisite, a high feeling of responsibility for psychologic reaction must be developed and finally the mundane factor, expense of equipment and meager financial return in relation to expended time and effort, must be considered.

The Army and Navy and Veterans Bureaus are primarily concerned with the profoundly deaf, that group which is not amenable to aids of hearing, which depends upon lip reading and speech training for its social and economic rehabilitation. The otologists' chief concern are those soldiers who are discharged or resign from the armed forces with a handicapping hearing loss. It may be assumed that all surviving combat aviators will be in this group — innumerable men who have been exposed to the incessant acoustic trauma of gun fire of all types. Regardless of the cause, the forgotten man will be the one with a loss ranging from 35 to 60 decibels in the speech frequency range.

At the present time the individuals whose hearing losses are in the upper level of this group, seriously handicapped as they are, do not fit into the rehabilitation scheme. They do present an important challenge to all otolaryngologists. We must be prepared to advise intelligently as to the pros and cons of hearing aids.

TYPES OF HEARING LOSS

Fundamentally there are three types of hearing loss for every level of impairment. Beasley's Public Health Survey[1] has merely confirmed on a grand scale what has been common knowledge to every otologist for many years. He has reported subjective disability as related to degrees of hearing loss. He[2] has further made recommendations for hearing aids as related to these specific losses. Theoretically these recommendations are sound. Practically, they are of no value because at no point is the psychology of deafness considered. This particular factor is the essence of all compensation by hearing aids.

Hearing losses are either uniform in degree throughout the audiometric range, the loss is accentuated at the higher frequencies (nerve deafness), or the low frequencies are most seriously impaired as in chronic middle ear suppuration. These factors are all modified by the duration of impairment, degree of impairment and type and by the age, sex and social condition of the patient. A hard of hearing or deaf person will always be an individual. He can never be treated merely as a member of a group.

HEARING AIDS

For practical reasons the present discussion will be concerned only with vacuum tube hearing aids of both air and bone conduction type. Approximately 25 such aids have been accepted by the American Medical Association's Council of Physical Therapy. For the most part unaccepted aids would not be generally used for the very reasons that they were not approved. However, the otologist should be familiar with hearing aids contained in the accepted list and see to it that his patient is safeguarded.

Modern vacuum tube hearing aids are fairly uniform in construction and design, the principle difference being in size, weight and appearance; also in size and weight of batteries.

The essential parts, all miniature, consist of a microphone, an amplifier or transmitter with two or three midget vacuum tubes, condensers, resistances, etc., a receiver which attaches to a fitted ear piece and a battery pack consisting of an A battery of 1½ volts and a B battery of voltage ranging from 20-45 volts depending upon the type and power of the instrument.

The amplification suppiied by the aid affects the audiometric frequencies involved principally in the speech range, namely 256-4096. By modifications in design and construction a certain degree of selective emphasis can be developed. This is usually in the higher frequency range. Low frequency amplification if modified at all is usually in the direction of suppression. Aids are available with a fixed frequency characteristic or are equipped with a tone control which modifies to a certain extent the character of the instrument's response.

THE FITTING OF HEARING AIDS

The fitting of the hearing aids must be a function of the otologist. It is essential, therefore, that the members of this medical specialty acquaint themselves with the intricacies, and the word is used advisedly, of this relatively new adjunct to their armamentarium. This is no easy task but it is an essential one and at the present must be adequately

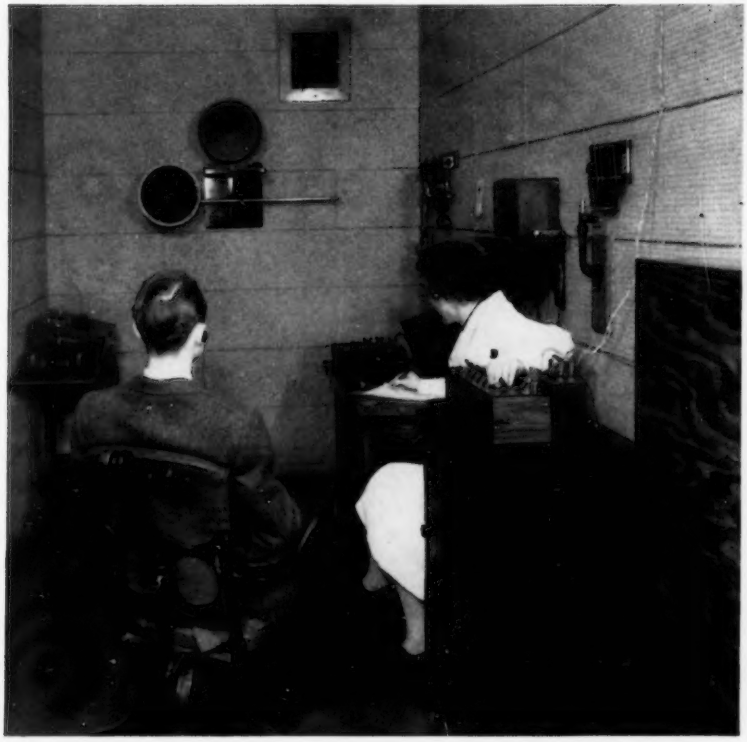

FIG. 1.* Hearing aids are tested in a sound proof room. The patient faces the loud speaker at a fixed distance. As the sentences are delivered the examiner controls the loudness level by means of the attenuator at her left. The speech reception threshold is determined first without an aid and then with one as illustrated. The second loud speaker is used to introduce measured intensity of a mixed noise during the speech reception test with the hearing aid if desirable. In this way actual working conditions can be demonstrated directly to the patient.

met. Space does not permit here of any discussion of the problem other than the actual methods of fitting hearing aids. The deaf or hard of hearing individual is concerned only with an instrument which will increase his ability to understand speech. The majority of acoustically handicapped persons are able to hear speech as delivered but it is meaningless to them. Therefore, an adequate hearing aid must supply sufficient compensation for a given acoustical deficiency to restore the vital quality of intelligibility. All other factors are essentially irrelevant.

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SPEECH RECEPTION TESTS

In this discussion the establishment of hearing levels based on uncontrolled spoken voice and whisper tests will be immediately dismissed as inadequate, unreliable and in the case of compensation utterly unfair. Such a test can only be of the slightest scientific consequence when the spoken voice is presented to the listener in the same degree of controlled intensity as are individual frequencies by the properly calibrated audiometer. Here again as in making discrete frequency audiograms a soundproof room is the ideal situation. If unavailable, a sound treated room should be provided.

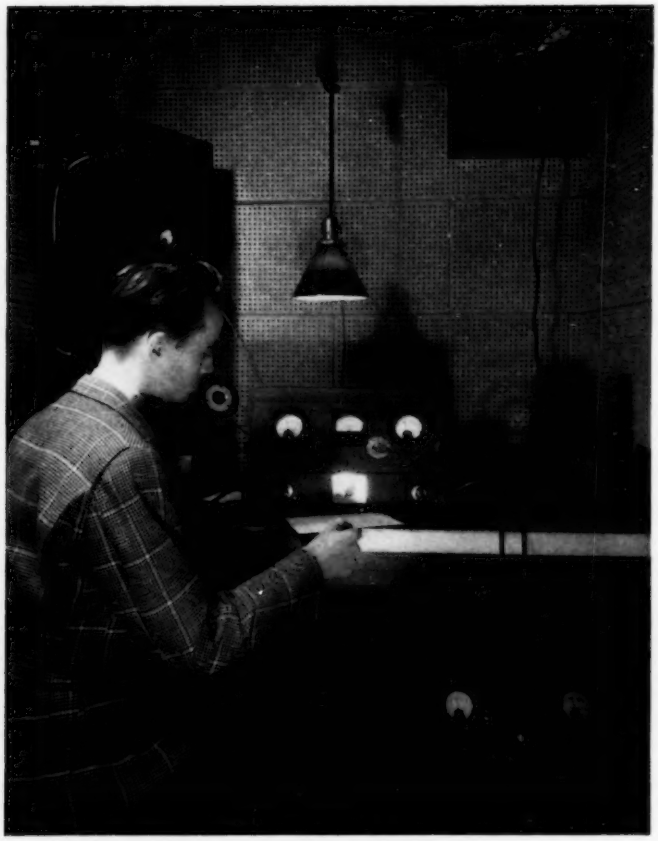

FIG. 2.* Sound proof booth in which reader speaks lists of sentences into microphone. Loudness of voice is controlled by decibel meter in amplifier.

Method

Three methods of presentation of spoken voice are available. First, spoken voice controlled electrically with an accurate attenuating system and presented indirectly to the listener by means of a high fidelity loud speaker.[3] Second, spoken voice presented through an electrically controlled circuit but using phonograph records as the sound source. Third, spoken voice presented through a roughly calibrated, non-electrical attenuating system, the general intensity levels of which can be reasonably well estimated.

1. This method requires equipment which will be found in only a few medical or acoustic laboratories. A soundproof room (Figure 1) is one of the essentials where speech can be presented to the subject by means of a loud speaker. The intensity of voice must have a calibrated attenuating control capable of modifying loudness in steps of five decibels or less over a range of 50 decibels above normal threshold. Beyond this point most electrical equipment tends to overload, and distortion is introduced.

A list of sentences is read by a speaker in a soundproof booth (Figure 2) through a microphone and an amplify-

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ing system. The intensity of the speaker's voice is modulated by observing the excursion of a decibel meter indicator needle. As in standard audiometry, the subject records accurate understanding of speech by establishing a level of accuracy of at least 60 per cent. Here accuracy is determined by repeating the presented sentences verbatim instead of pressing the signal for heard or not heard as in the audiometric examination.

Sufficient time is allowed between the presentation of each sentence for the subject to repeat what he has heard without undue hurry. Each sentence has a key word or words, which are critical in the correct repetition of the sentence. Interestingly enough this method is accurate to plus or minus one decibel and does not introduce any element of deterioration in production and is completely flexible, both of which factors must be taken into account in method two.

2. Phonograph records are used in this method for the speech source, but the general electrical system and physical surroundings are essentially the same. This method requires one less in the examining personnel but is entirely inflexible as to timing and immediate choice of sentences presented and is highly susceptible to record deterioration due to repeated playing. The sentence lists used are those devised by the Bell Telephone Laboratories and comprise 50 lists of 50 sentences, each of which would involve a very considerable number of records.

3. If none of the above equipment is available a reasonably accurate test can be carried out in the following manner. This test is based on the assumption that the normal spoken voice of any individual is more accurately controllable than an attenuated voice such as whispering.

Any standard two-foot megaphone increases the intensity of voice spoken into it by approximately 20 decibels. If normal speech is of the order of loudness of 40 decibels above threshold and to this is added an additional 20 decibels, few impaired hearing levels susceptible to artificial aid would fail to be reached through such a system. Attenuation in the system can be introduced by a series of felt discs graded in diameter to that of the megaphone at the point of their placement within the megaphone. Such a system can readily be constructed so that each felt disc will introduce an approximate attenuation of 10 to 15 decibels. If four discs are placed, the total loudness range of the device is 60 decibels. The speech reception level is first determined by establishing the distance from the subject at which the spoken sentences are repeated accurately 60 per cent of the time without any of the discs in place. This distance is recorded. The mouthpiece of the megaphone must be circled with some type of sponge rubber which will prevent escape of sound outside of the megaphone. (Figure 3.)

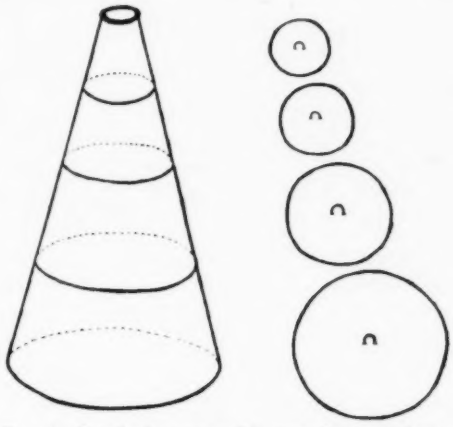

FIG. 3. Standard commercial megaphone 2′ long. Discs are spaced 6″ apart and introduce attenuation of approximately 12 decibels for each disc giving a total attenuation for spoken voice of 40 decibels. The felt is standard ½″ material stiffened by means of wire screening.

FITTING THE AID

In any one of the three systems described, an unaided speech reception level is determined. Air and bone conduction audiograms have been made, and otological examination has determined whether or not the impairment is complicated by a chronic suppurative otitis media or not. In general this is the prime deciding factor between the use of an air or bone conduction hearing aid.

Hearing aids are either constructed with a specific frequency characteristic, flat, high frequency or low frequency emphasis or have tone controls which change the characteristic of the instrument to fit the specific need. In addition, with several aids receivers are supplied which are supposed to contribute to intensity as well as frequency response. If the receiver is a crystal one, the larger the receiver the greater will be the high frequency emphasis particularly in the neighborhood of 3000 cycles. This is an important factor.

It is quite natural that an instrument complementing the audiometric hearing loss would be the one first chosen for test. However, this may very well not be the one finally selected. The known overall gain for speech must be a matter of record.

Whichever of the three testing methods listed is used the extent of gain over unaided hearing will be the measure of each individual instrument's effectiveness. By the electrical methods the addition of attenuation is the measure of acoustic gain; by the megaphone method the number of felt discs which can be introduced into the device and still have speech intelligibility will give a rough estimate of the aid's amplification.

Many factors are involved in this problem. In the war casualties, advanced age (the most difficult group of all to fit) will not be a factor. The serviceman will not be particularly concerned with the appearance or size of the instrument and batteries so long as the desired results are obtained. These factors are often critical in the fitting of aids for civilians and particularly, of course, women.

But in the two groups there is one sine qua non. No hearing aid of either air or bone conduction type should be considered unless it gives a gain of 30 decibels in speech reception regardless of the established unaided hearing level.

As an addendum it can be stated that vacuum tube aids are vastly superior to carbon microphone instruments. Air conduction aids are basically far superior to bone conduction aids which represent a hearing efficiency under optimum conditions of 30 decibels below normal air conduction and which have a frequency range limit of less than 3,000 cycles, which is the critical level for normal speech reception.

A bone conduction aid should never be prescribed if it is possible to use an air conduction instrument.

EAR MOLDS

For air conduction a well fitted ear piece is essential. Even with the best instruments the "feedback squeal" will develop if the receiver and earmold fit is not an adequately close one. Many hearing aid agencies are equipped to make plaster casts of the external ear and cartilaginous canal. Dentists also perform this service. The latter are undoubtedly better equipped provided the otologist has made an otoscopic examination and can give a satisfactory report on the patency of the external canal and the intactness of the tympanic membrane. Any ear mold made without such supervision is a direct, specific and unauthorized assumption of a medical prerogative and should be susceptible to action under the established legel rules of medical practice.

SUMMARY AND CONCLUSION

The present world situation poses two major problems for the otologist; first, the immediate relief of civilian hearing impairments as an aid to the war effort, and, second, the care and rehabilitation of aural casualties of the war who are returned to civilian life. It is true that many of these veterans will still be under the direction and control of the Veterans Bureau, but there will be a highly significant number who for some reason will be without medical advice and control. American otologists must assume this responsibility in the broadest possible manner. They must familiarize themselves with the construction and performance of Council Accepted Hearing Aids. They must conform in their technic of fitting aids to some more or less uniform standard of procedure. Their records of successful performance will be a matter of great importance as will be their record of failure.

The medical prerogative in this relatively new field should be jealously guarded. Hearing aids are manufactured and sold primarily for profit. Variations in sales technic and accepted advertising methods are designed to increase this profit through greater volume of sales. Beyond the fact that their product is an excellent one, the hearing aid companies should have no particular obligation or concern. We have been laggard in assuming our responsibility in this very important part of the therapy of deafness. That we must assume it now permits of no discussion.

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DISCUSSION

JOHN J. SHEA, M.D., Memphis, Tenn.: This presentation of Dr. Walter Hughson's is authentic and should be a stimulant to us. The members of the Academy who practice the combined specialty will make a better ophthalmological examination than an otological examination. No one practicing ophthalmology would just tell his patient to take his prescription and buy himself a pair of glasses. He is definite in what he tells him. Still that same specialist will make an incomplete otological examination, and then tell the patient to buy a hearing aid. There is a reason for that. I believe that men who practice otology are not as well trained to make otological examinations and are not as well equipped in their private practice as they should be. This is my opinion as an examiner on the American Board of Otolaryngology for fifteen years.

It took a century for optics to develop to the point where a prescription could be filled, but out of all the scientific work being done upon noise in this present war there may come out of it better hearing aids, and in the future we will not only write a prescription for our hearing aid but we will get one for the individual.

In this connection, there is no question that light affects many eyes that are not pathologic. The same thing holds with ears, and in some individuals who wear hearing aids noises produce headache. This is a phase that none of us has investigated.

I believe that the otologist should decide whether a carbon set or a vacuum set is indicated. He should also decide whether an air-conduction apparatus or a bone-conduction apparatus is needed and to which ear it should be fitted. That is our responsibility and there is no way for us to avoid it.

I want to congratulate Doctor Hughson on his presentation.

G. F. HARKNESS, M.D., Davenport, Iowa: It is a pleasure to be asked to discuss Doctor Hughson's paper. We are fortunate in the medical profession to have men such as Doctor Hughson giving their time and effort in an otologic research laboratory. The fundamentals that may be established in such work are basic in the clinical application of our knowledge to the relief of the handicapped

individual. If I am to disagree with some of his conclusions they are presented as from one who is on the firing line of clinical otology, the practicing physician.

It is true there are many times the number of men in Aviation in this war compared to those in World War I, but the total of our armed forces is not six times the number of those in World War I. The assumption that no combat aviator will return to civil life with normal hearing is an unwarranted assumption without statistical support. Likewise, to include as a prominent factor the remote effects of serum and antitoxin administration and prophylactic drug therapy as causes for aural casualties cannot be accepted without scientific proof. It is to be hoped that the author has overestimated the actual aural casualties that will result.

Hearing aids are part of a rehabilitation problem. Rehabilitation is not a simple medical and scientific problem. It is also an economic and industrial one. We can establish standards for specific jobs, but as jobs vary so will standards vary. Otologists in the past have been woefully lacking in their interest relative to the problems of the deaf. The hearing aid is not the product of the brain of the medical otologist but rather of the physicist and the acoustic engineer. In fact the otologist cannot with finality even tell the physicist how we hear. We have become decibel conscious, but loudness is measured not in decibels but in millewatts. With every tone frequency there is a definite intensity necessary to make the tone audible. The main problem of the deafened individual is the intelligible perception of the human voice as a means of communication. The human voice is a mixture of tones, the perception of vowels lies between 500 and 2,500 cycles, the voiced consonants between 1,000 and 3,000 cycles, and the unvoiced consonants between approximately 2,250 and 8,000 cycles. The percentage scale for syllable articulation as developed by Fletcher and Steinberg by which we can evaluate the effectiveness of the various types of amplification is a practical avenue of approach to the perception of the human voice. These tests as prescribed are conducted in a sound proof room, but neither you nor I or the deafened individual carry on this perception in a zero noise level environment. Furthermore, the surrounding noise level in which you work may differ from mine or of the deafened individual. Are these factors of moment in the hearing aid?

The auditory sensation area in typical conductive deafness is approximately the same size as that of the normal ear, the threshold for hearing and feeling being both raised. In perception deafness this area is reduced because the threshold for feeling remains practically normal. The hearing aid is a means of amplifying sound. Uniform amplification does not bring all speech frequencies to the same level but amplifies each tone by the same amount. With the sensation area practically normal in conductive deafness the carbon hearing aid is of great benefit to this group.

With the miniature vacuum tube amplifier it has been possible to vary the amplification at different speech levels, so-called selective amplification. This has mechanical limits of application and limits of effectiveness because of the type of hearing loss of the individual. Selective amplification disturbs the usual relative speech sound levels. This often means adaptation and accommodation by the individual to the aid. Again the hearing loss at the threshold of hearing does not remain constant with an increase of intensity. Furthermore, the hearing loss at the most comfortable level for speech perception may be greater than at the threshold of perception. This is more marked in the perception type of deafness. When the hearing loss for speech approximates the most comfortable level, uniform amplification is most successful. Otherwise selective amplification should be prescribed.

I am mentioning these factors that constitute but a few of those that add to the complexities confronting hearing aid prescriptions. I find myself not agreeing with the author in some of his conclusions. You can drive an automobile and not know what is under the hood. To justify the prerogative that the prescribing of hearing aids be exclusively ours as a part of our therapeutic armamentarium, we must qualify for the job. I am viewing this from the standpoint of the private practicing otologist who must earn his sustainance almost by the sweat of his brow or what he has back of it. We must know why the aid does or does not give linearity of performance. Non-linearity means overtones and masking of original sounds with distortion. We must learn the causes of adventitious noises, of static and the like. We must learn the reasons for stability or lack of stability from microphone to ear phone. We must learn the limits of tone and pitch selectivity in the carbon aid by changes in combinations of diaphragms. In the vacuum tube aid we must in addition learn the results of alterations in the vacuum tube amplifying circuit. We should understand why the carbon and why the crystal microphones should be prescribed. We must learn

that certain substances as Rochelle salt crystals exhibit electrical charges when subjected to pressure and how that affects their action in a microphone.

Audiofrequency waves which are attached parasitically to powerful oscillations of radio frequency are shunted off in the receiving set, amplified by means of the vacuum tubes and then attenuated to a level acceptable to the sense of hearing. The hearing aid likewise amplifies to its maximum power of audibility to have this maximum power attenuated, short circuited if you please, to the needs of the individual. All these, if the prescribing is to be the otologist's province, he must master. In this question of who fits the hearing aid must be considered economic factors and the most good to the greatest number.

I agree with the essayist that the otologist should become familiar with approved hearing aids. He should acquire sufficient knowledge to advise his patient wisely. I agree that the psychologic factor is a most important one. The otologist should be master of this. The business man calls it sales resistance. Convincing the patient that the wearing of a hearing aid is no stigma has long been the first barrier to hurdle, and I cannot agree when the essayist says that the ex-service man will not be particularly concerned with the appearance or size of the instrument so long as the desired results are obtained. Let us face facts. Let us not adopt a holier than thou attitude. We do have glass in our own house. Let us not assume the right to a medical prerogative unless we can justify it by the services that the rank and file will deliver. This will be measured by the patient's sense of value received for his money expended.

Of course hearing aids are manufactured for profit. There is a legitimate research expense, there is a necessary high legitimate selling expense, but there are manufacturers with high ethical standards who do feel an obligation beyond increased sales. There have been and will be others who are charletons, just as we have them. It is our duty to know who is who.

Could the acoustic laboratory under medical supervision accommodate all those needing hearing aids then I would agree that this is a medical prerogative. But I feel that this is a laboratory dream. Will practicing otologists as a group perfect themselves with all knowledge necessary to fit hearing aids successfully, will they give the necessary meticulous time-consuming examinations and will the average hard of hearing individual be willing to compensate adequately the otologist for his time? To these questions I believe the answer is "No." The past lack of interest by otologists in the problems of the deaf only supports the negative. Without adequate compensation I can even visualize an abuse of such a medical prerogative, where even some men might forget their Hippocratic oath and be susceptible to a hidden compensation. The pitfalls of misplaced confidence above everything else are to be avoided.

Several years ago during the carbon aid period, with the cooperation of a number of manufacturers I had their instruments installed in my office. I found it impractical. There was not enough demand to justify the employment of a skilled technician, and I could not give it the necessary time. With selective amplification the difficulties would have been multiplied.

The author's ingenious method of megaphone testing of the aid's amplification relative to speech intelligibility is, as he states, a rough estimate. No doubt it is very practical as he uses it, but because of variations of examiners, it would seem to lack something in standardization, to be adopted for general use.

Let us give credit where credit is due. The hearing aid is the product of the physicist and acoustic engineer, not the otologist. Let us be so familiar with it that we can advise wisely. Let us cooperate. Let us avoid any taint of commercialism. Let us realize that there is no utopia we couldn't attain if given carte blanche but we can strive to help the most people in the most practical way.

I think the Academy is to be congratulated in selecting Doctor Hughson to present this paper and he on the fine presentation of the same.

WALTER HUGHSON, M.D., Abington, Pa.: I should like to thank Doctors Shea and Harkness for taking the time to discuss this paper.

I should like to add something to Doctor Shea's remark that optics are a century old, indicating, as I know he meant to indicate, that the use of hearing aids is in its infancy. I believe the first picture of an individual with glasses was painted in 1200 A.D. The first vacuum tube hearing aid was made approximately 15 years ago.

The noises produced by ill-fitted hearing aids, which both of my discussers mentioned, of course, I am sensitive to, as is every patient. It has been impossible in this brief discussion to describe the refinements of technic which must be developed in fitting a hearing aid, such as presenting speech to the patient

in a noise field comparable to that in which he may have to work.

I hope very much, as Doctor Harkness has said, that my statement in regard to the number of war aural casualties is greatly exaggerated. The Surgeon General's Office and several flight surgeons have stated specifically that they thought this number was an exaggeration. However, on the basis of returns from the last war, my estimate, although large, is not out of reason.

In discussing in detail the structure of hearing aids, naming the integral parts and describing the performance of the instruments, Doctor Harkness has supplied information which the scope of my paper could not include. I am pleased to have this data be a matter of record. Doctor Harkness feels that otologists should not be expected to know these facts, but he himself sets the example which we all should follow. He feels that hearing aids are products of the skill of electrical engineers and that otologists should consequently not assume responsibility for the application of the aids for the relief of hearing loss. I cannot agree with this feeling in any particular. Were there no such physical disability as impaired hearing there would be no hearing aids. In all other branches of medicine, special technics involving the use and application of special apparatus is common practice. My one and constant purpose will be to interest otologists in acquiring a satisfactory intelligent knowledge of the structure and use of hearing aids. If mistakes are to be made let the responsibility lie where it belongs — on us. We are practicing medicine and use an instrument to treat a disability. The electrical engineers manufacture hearing aids; they cannot practice medicine. I hope very much that otologists will take the time and trouble to learn the intricacies of the hearing aid problem. It is an obligation which we have no right to shirk.

INDUSTRIAL OPHTHALMOLOGY*

A VISUAL SERVICE FOR SMALL MANUFACTURING PLANTS AIMED AT THE PREVENTION OF BLINDNESS BY THE ELIMINATION OF INDUSTRIAL ACCIDENTS

HARRY S. GRADLE, M.D.
Chicago, Illinois

According to the best figures available, approximately 15 per cent of the blindness in the United States is the result of accident, fully four-fifths of which occur in the industries. Such industrial accidents may be due to poor vision on the part of the employee, to inadequate illumination so that visual hazards are not seen, or to the thousand and one other dangers.

This problem of blindness due to industrial accident has never been approached in a methodical manner, although the majority of the larger industrial concerns maintain a medical and a safety department, which is more or less efficient in this matter. However, in the smaller plants whose size precludes the maintenance of a medical staff or of a department of safety, the visual hazards receive but little notice. Consequently the Illinois Society for the Prevention of Blindness deemed it a part of its duty to try to aid the smaller manufacturing plants in the elimination of the various visual hazards that all too frequently lead to blindness.

Therefore, a service was devised and is now in operation, a service primarily for the smaller manufacturing plants. By that is meant the plants with from a score up to 700 or 800 employees. This service operates in the following manner:

1. The management is contacted and given full details of the service, and if they accept it, a contract is signed.

2. A "tester" contacts the various foremen and arranges a routing schedule whereby the employees are sent to the selected place in the plant on such a time schedule that there is a minimum interference with the normal operation of the plant.

3. At the appointed time the tester conducts a visual survey of each employee, using the new Bausch and Lomb Orthorater. With this instrument is measured:

a. The visual acuity of each eye for distance, without glasses.
b. The visual acuity of each eye for distance with the employee's glasses.
c. The visual acuity of the two eyes combined for near, without glasses.
d. The visual acuity of the two eyes combined for near, with glasses.
e. Muscle balance.
f. Depth perception.
g. Color sense.

This is all recorded on a special form on which is noted the employee's name, age, sex, occupation and department of the plant in which he works.

4. While the visual survey is being conducted, a trained safety engineer makes a survey of the entire plant, including the offices, in which is taken into account:

a. The general illumination.
b. The illumination of individual pieces of work.
c. The illumination both by daylight and at night.
d. The visual hazards that exist in each portion of every operation of the plant, both from the standpoint of flying particles, dangerous obstructions, dust, etc.

In this is considered not only the operator of every individual machine, but also the safety of passers-by.

5. The report of the vision tester is then analyzed from a medical standpoint, department by department, and is submitted to the management. In that are recorded the names of the employees who require correction of visual defects, who have a manifest ocular disease, who have a marked imbalance of ocular muscles, who are deficient in color sense, or who are deficient in depth perception. The urgent cases are starred.

Eleven minutes is the average time required per employee for this visual survey. The office personnel with higher intelligence require about 7 minutes per individual, whereas those of the laboring class, often with a deficient knowledge of

* The following papers on Industrial Ophthalmology were presented at the program sponsored by the Committee of Industrial Ophthalmology during the Forty-Eighth Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 10-13, 1943, Chicago, Illinois.

English, require up to 18 minutes per individual. In the medical report the management is urged to make sure that the employees who have visual deficiencies seek medical aid and receive the necessary correction. The original plans for this service included refraction by a certificated ophthalmologist at the plant itself; but this had to be abandoned because of the shortage of ophthalmologists. Another proposed phase was to have a reputable optical concern fill those prescriptions for glasses or protective goggles at the plant. But here, too, the manpower shortage has necessitated abandonment of that part of the program.

Attached to the medical report is a list of members of the Chicago Ophthalmological Society who are not in military service. The management is urged to avail itself of the services of any of the men who are listed.

6. The report of the safety engineer accompanies the medical report. In it are described the areas of insufficient illumination, together with recommendations as to the type of fixture and the wattage necessary to correct the defect. In it are described the various visual hazards from operations within the plant and the type of protection requisite to eliminate such hazards. Those may be in the nature of a machine guard, protective screen, goggles, or whatever the exigencies of the situation may demand. These also are prepared department by department.

7. The Society also offers the services of members of its staff to promulgate a safety campaign among the employees of the plant. That campaign is in the nature of posters, brochures on eye safety, moving pictures of industrial accidents and their consequences, first aid directions, etc.

For these combined services the management pays the Society 50 cents per employee. This, of course, does not cover the cost, and a special fund has been earmarked in the treasury of the Society to continue this as requisite. As time progresses the various plants will be visited again by members of the staff, and further endeavors will be made to see that the managements comply with the recommendations of both medical and engineering reports.

Although this service is new, it is believed that much will be accomplished in the prevention of blindness from industrial accidents.

HOW CAN OPHTHALMOLOGISTS BE OF GREATER SERVICE TO INDUSTRY IN WARTIME?

C. D. SELBY, M.D.
BY INVITATION
Detroit, Michigan

The ophthalmologist serves industry as a consultant in the handling of occupational injuries of the eyes and as an expert in the settling of claims for compensation involved in eye injuries.

Industrial medicine began in a small way with the care of occupational injuries. Over the years this has expanded into a complete health program in which the care of injuries, though still important, is relatively a minor function.

From the title assigned to me for discussion it is assumed that some suggestions are wanted as to how the ophthalmologist can participate in the broadened industrial medical program and thereby extend his opportunities for service to the industries which are giving to the war effort. Let us work this out together.

What is the industrial medical program? It consists of those medical functions which concern the prevention of sickness and the care of occupational diseases and injuries. The objective is to control disabilities of occupational origin and give the employees the best possible health protection consistent with the purpose of industry, the employer's legal responsibilities and the employee's rights in the free choice of physician for care of sickness and injuries not legally related to occupation.

These functions are specified in the "Outline of Procedure for Industrial Physicians in Industry" which was prepared by the Council on Industrial Health of the American Medical Association and published in the March 14, 1942, issue of the Journal. I shall quote from this and after each section let us consider the possibilities for ophthalmological assistance and guidance to the industrial physician.

"**Prevention**—The physician should acquaint himself by regular inspections with all materials and processes used in the working environment over which he has supervision, to the end that he may recommend appropriate protection of employees from conditions actually or potentially harmful."

Suggestion—This suggests the possibility of studies of the illumination of manufacturing areas, the kind of lighting

that is best suited to the different occupations, the placement of fixtures and the projection of the light for best results with a minimum of fatigue to the eyes of the workers.

"Industrial Physical Examinations — Preplacement physical examinations should be complete. They should be used only for the purpose of assigning work adjusted to the physical and temperamental fitness of applicants and to maintain safe and healthful employment for all employees.

"Subsequent physical examinations should be complete enough to provide positive health protection for all workers and to safeguard the public welfare. Repetition of such physical examinations must be determined by the physician in accordance with specific requirements.

"In the interest of completeness and uniformity, physical examination forms are recommended. Personal records of this character are confidential and should always be kept in the custody of the medical department. Access to these records should be granted only on request or consent of the examinee.

"The examining physician should acquaint the examinee with the results of all examinations or take steps to refer all conditions requiring correction to the physician of the worker's choice."

Suggestion — This suggests participation in the examination procedures. As you know, so far as eyes are concerned industrial physicians are general practitioners. Their examinations of eyes are necessarily superficial. They may use the ordinary Snellen chart or one of the devices for testing eyes. To some degree they check near vision and possibly the glasses that are worn though, admittedly again, superficially. Unless a workman is unsighted the quality of his production depends substantially upon his eyesight—usually his near vision. Why not propose a method of participation in the examination of the eyes and the glasses of the workers with the view to obtaining correction where needed and proper job placement?

"Health Education — The plant physician should take advantage of all opportunities for beneficial instruction of the workmen in hygienic living both in and out of the industrial environment."

Suggestion — This suggests opportunities for advice to employees on the care of their eyes. Possibly talks on the use and health of the eyes, etc.

"Medical and Surgical Care — 1. Treatment of Compensable Injuries and Diseases: The disabled worker should be free to choose his physician from all those licensed doctors of medicine competent to supply the required services except in situations provided for by chapter III, article VI, section 3 of the Principles of Medical Ethics. This provides that the intervention of a third party who is legally responsibile either for the cost of care or for indemnity or who voluntarily assumes partial or full financial responsibility for medical care does not per se cause a contract to be unethical.

2. Treatment of Noncompensable Injuries and Diseases: The treatment of injuries or diseases not industrially induced is the function of private medical practice. The physician in his industrial relationships should abstain from such services except in the case of:

A. Minor ailments. The physician in industry may treat minor physical disorders which temporarily interfere with an employee's comfort or ability to complete a shift, and for the relief of which he may need immediate medical attention.

B. First aid for urgent sickness. The physician in industry should employ such measures as the emergency dictates in all cases of urgent sickness occurring during working hours on the working premises, until such time as prompt notification of the family physician relieves him of further responsibility.

C. Rehabilitation after sickness and injury. The physician in industry can properly assume responsibility for those phases of rehabilitation after disability industrially induced or otherwise, which can be best controlled under working conditions."

Suggestion — It is evident that the medical and surgical care of eyes is limited in industry and no services in this respect are necessary beyond those which the ophthalmologist now renders. Nevertheless, it might be possible to make arrangements for on-the-job service which would entail at least a part-time attendance at the plants. This is practiced in some large industries with good effect.

Research — In the group of plants that I know best, certain obscure eye problems, which do not appear to be covered in the procedures advocated by the Coun-

cil on Industrial Health, arise from time to time. For instance, some occupations, such as welding, may be thought to be harmful to the eyes. This question was put to our doctor. An ophthalmologist was asked to make a study of the eyes of the group. He did so and his report was convincing to the workers and comforting to the management. These and similar problems do arise.

Suggestion — This suggests the need of industrial ophthalmological research. You may see possibilities in this of which industrial physicians are not aware.

Goggles — Here is a very practical matter. Goggles are used for eye protection. Workers will not wear them if they are ill-fitted and uncomfortable, if the lenses steam up, if they cannot see through them clearly and for many other reasons. So the expert fitting of goggles, their maintenance, sterilization and repair, and the use of prescription lenses where needed are at all times important. Industry has had difficulty in securing the help of ophthalmologists in these matters.

Suggestion — This suggests the need of a goggle program.

Concluding Suggestion — The suggestions thus far made have arisen out of practical experience. It may be possible for a group of ophthalmologists, whose contacts with industry are obviously casual, to comment upon them, improve them and offer others. But I am inclined to believe that the ultimate solution of large industries' eye problems is in this final suggestion, that part-time industrial ophthalmological service be advocated by your group and that measures be taken to acquaint the medical directors of large war industries with the potentialities of such a service.

THE USE OF VISUAL DATA AS AN AID TO INCREASE PRODUCTION AND EFFICIENCY

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During the past four years a number of experiments dealing with the relation between visual performance and employee efficiency on the job have been conducted.* From the data, it appears that certain **patterns** of visual skills are associated with high production on certain jobs, while somewhat different — or even markedly different — patterns of visual skills characterize the high producing employee on other jobs.

A brief discussion of what is meant by "pattern" of visual skills may clarify the preceding statements. The visual skill tests used are of several types. Some deal with visual acuity at 14 inches and at 20 feet. Each of the visual acuity tests is given as a binocular and also as a monocular test. Other tests measure the vertical and lateral postural tendencies of the eyes, while still others measure depth perception and color vision. In all, the tests used in most of the experiments include seven tests given at the optical equivalent of 20 feet and five given at the optical equivalent of 13 inches.

These tests may all be given conveniently and reliably in the Ortho-Rater — an instrument designed by the Bausch and Lomb Optical Company for the visual classification of employees. The Ortho-Rater is designed to be used in industry by specially trained lay testers. The tests measure only visual performance or visual skill. They have been carefully designed so as not to be diagnostic in nature.

Early in the course of the experimentation it became apparent that a person who scores very high on any one test may not be significantly above average on any other test. A person may be high on certain of the tests and average or even well below average on others. For example, a person may have very keen distance acuity, but only average or below average near acuity. The same person may score high in depth perception, and low in color discrimination. Another person may show a still different pattern with respect to any — or even all — of the tests. The pattern, of course, includes the phoria tests as well as those that have just been mentioned.

When the concept of a "pattern" of visual performance — rather than any single skill (such as acuity) — is used as the basic principle of analysis with which

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* This work has involved the collaboration of Purdue University, particularly S. E. Wirt, Ph.D., and other members of the staff of the Division of Education and Applied Psychology; H. S. Kuhn, M.D., of Hammond, Indiana; the Bausch and Lomb Optical Company, Rochester, N. Y.; and a number of industrial plants in which the tests were made.

to formulate the visual requirements of different jobs, many interesting and valuable results are revealed. For example, on one job that was studied — radio tube assembly — there was no significent relationship between distance acuity and job performance. The employees testing lowest in distance acuity — several as low as 20/40 when tested binocularly — were doing work that was as satisfactory in all respects as the work of the employees with the best distance acuity. In other words, if the vision tests had been limited to this one test, it would have been necessary to conclude that there is no relationship between vision and performance in the job. However, when analyses of the other vision tests were made, several showed a decided relationship with successful performance on the job. Charts showing certain of these results will be presented later. For the present, it will suffice to say that only by thinking in terms of the concept of a "pattern" of visual performance was it possible to discover the true relations existing between vision and job performance. Once this concept has been accepted and the production or quality of employees' work checked against the pattern of their visual performance, relationships that had been entirely unnoticed often become strikingly apparent.

It is to the advantage of both management and employees to discover these relationships, to place new employees on jobs for which they are visually qualified and to obtain professional service for those applicants or employees whose visual performance does not conform to the pattern which characterizes the high producing employees. The procedure followed in determining the optimal visual pattern for each job consists in giving the battery of vision tests to both high producing and low producing employees on that job, and determining statistically those vision tests which differentiate between high and low producing operators.

The Ortho-Rater furnishes a convenient and rapid means of administering visual performance tests that have been found to correlate with successful job performance on a variety of industiral jobs. By means of the perforated record cards used with the Ortho-Rater, it is a simple matter to determine, for any job, which vision tests correlate with satisfactory job performance and which ones do not. After this has been done, the production efficiency of employees may be plotted as a statistical function of their test scores for each of the tests under consideration. These plotted curves clearly reveal the test were superior (rated "A" or "B") operators on the job.

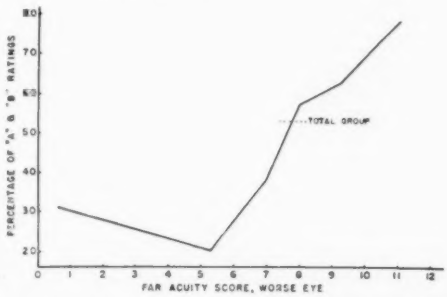

FIG. 1 — The relation between job performance on piston ring inspection and distance acuity test score (worse eye).

Figure 1 shows such a curve for distance acuity (worse eye) plotted against efficiency of the operators on one job — inspecting piston rings. The employees were divided on the basis of production ratings into "A," "B," "C" and "D" operators. Fifty-three per cent of the total group were "A" or "B" operators, and the remaining 48 per cent were "C" or "D" operators. Figure 1 shows that there is relatively slight change in the percentage of "A" and "B" (high producing) operators between scores of 1 and 5 on the distance acuity test (worse eye), but that for higher test scores there is a marked increase in percentage of superior operators with each increase in test score. This increase is so great that approximately 80 per cent of employees scoring 11 on this test were superior (rated "A" or "B") operators on the job.

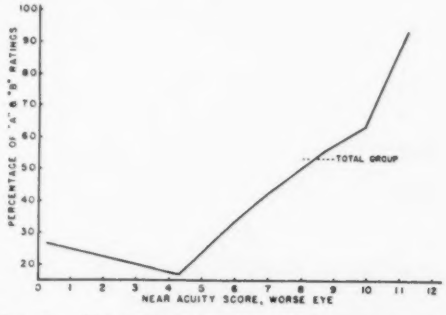

FIG. 2 — The relation between job performance on piston ring inspection and near acuity test score (worse eye).

Figure 2 is a corresponding curve showing the relation between near visual acuity (worse eye) and percentage of superior operators. As in figure 1, the relationship is not significant in the lower half of the range of test scores, but is marked in the upper half. Indeed, the relation is more marked than in the case of distance acuity, for 96 per cent of the employees scoring 11 on this test had been identified as superior operators.

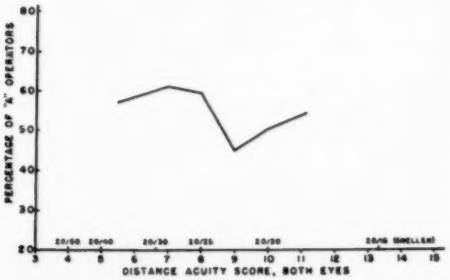

FIG. 3 — The relation between job performance on radio tube assembly and distance acuity test score (binocular).

Figures 1 and 2 show that for this particular job of piston ring inspections high scores on both distance and near visual acuity tests are required for superior performance on the job. The fact that high acuity in the **worse** eye is required means, of course, that high acuity in **each** eye is necessary. Not all jobs, however, demand both these characteristics. Figure 3 shows the relation between distance acuity score and percentage of superior employees among radio tube assemblers. It will be noted in this case that there is no systematic relationship between tests scores and efficiency on the job. Employees with low scores on this test are rated "A" as frequently as those with high scores.

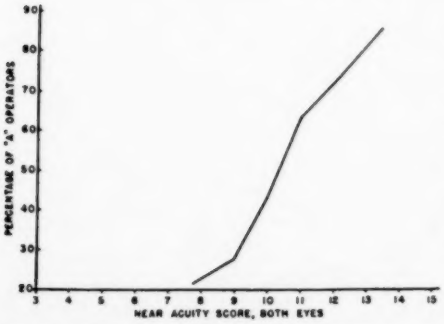

FIG. 4 — The relation between job performance on radio tube assembly and near acuity test score (binocular).

Figure 4 shows a markedly different situation when production ratings are plotted against **near** point visual acuity. Only 22 per cent of the operators with a score of 8 on this test were rated as "A" operators, while 90 per cent of those with a score of 13 were rated as "A" operators. For successful performance on this job, keen distance acuity is not needed, but keen acuity at close distances is highly important.

Analyses of this type made in the plant include all of the other Ortho-Rater tests as well as those for near and distance acuity. From these test results, a visual profile for the job may be readily constructed. Such a profile shows within what limits on the several visual performance tests an applicant should score if he is to get along well on the job in question. Those who do not fall within these limits should ordinarily be placed on other types of work for which they may be well qualified visually, or they should be referred to an eye physician for attention. By following one or the other of these procedures, practically every applicant, no matter what his visual performance may be, can be salvaged for some type of work in the plant. The visual requirements of different jobs vary so widely that practically all able-bodied men and women are visually qualified for **some** type of work.

The advantages of this program are numerous. It is frequently possible to increase either quantity or quality of production by amounts varying between five and 25 per cent. Such an increase is of value both to management and employees, particularly when a wage incentive plan is in operation. Learning time is often shortened for new employees because they can adapt more readily to a job that does not call for great variation from their present visual performance. Eye strain and fatigue are reduced for the same reason.

The success of this program is largely dependent upon cooperation between personnel and industrial relations men, on the one hand, and the profession of ophthalmology, on the other. Your profession has a real service to render industry in helping to "fit the eyes to the job," and the evidence we have been gathering at Purdue shows that this service will pay real dividends to management, employees, and to ophthalmology.

A CROSS SECTION OF EYE SERVICE IN INDUSTRY*

CHARLES P. TOLMAN
BY INVITATION
Consulting Engineer
National Society for the
Prevention of Blindness

I greatly appreciate the honor of addressing this body. I thank you for the opportunity on behalf of the National Society for the Prevention of Blindness which I represent as Consulting Engineer. I speak with a deep sense of responsibility because I must not fail to convey to you our findings on the serious deficiency in practices affecting eye conservation in industry and the effect of this deficiency upon the war effort.

As a "general practitioner" in engineering, I have come into contact with many industrial eye lesions as a result of ocular deficiency, if I may apply your terms to engineering work. I do not hesitate to say that none were so serious or so far reaching in their bad effects on industry as the lack of knowledge and concern about the conservation and use of vision in industry.

In ordinary times a considerable part of the loss might perhaps be ignored from the professional viewpoint as economic losses not directly involving humane considerations. However that may be, the war has made the entire subject a direct professional responsibility. No professional group can do so much to correct the deficiencies and thereby improve war production as ophthalmologists.

The engineers have done their utmost in methods and equipment. Others have made secure the supply of raw materials. This equipment is being operated and these raw materials are being used by workers of whom we believe at least one in every four cannot properly see what he is doing.

Several members of your profession have contributed notably to the solution of the many problems of industrial eyesight. By unremitting study in the plants, they have applied ophthalmology to the job and have developed practical methods, basic principles and technics for the solution of these problems.

Your profession is also fortunate in having the cooperation of technical concerns, on a fine ethical basis. Through such highly skilled service, instruments of outstanding importance to the industrial program are being developed.

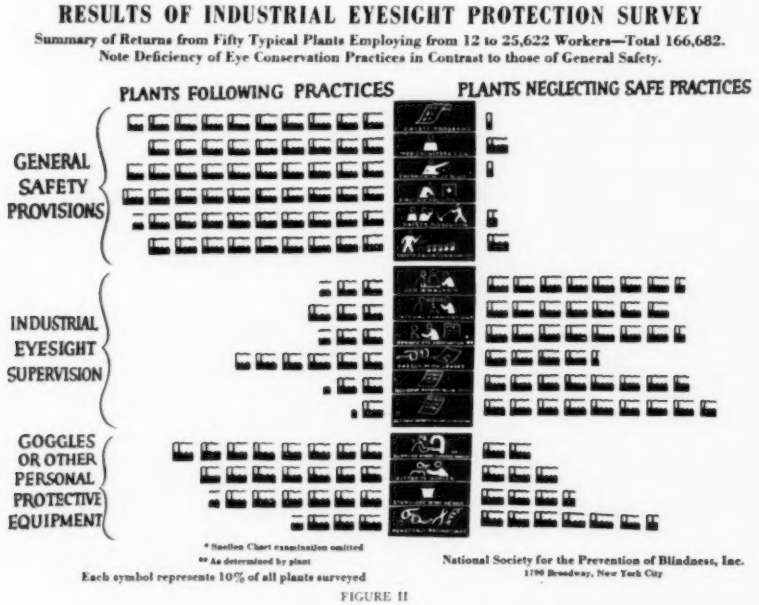
RESULTS OF INDUSTRIAL EYESIGHT PROTECTION SURVEY
Summary of Returns from Fifty Typical Plants Employing from 12 to 25,622 Workers—Total 166,682.
Note Deficiency of Eye Conservation Practices in Contrast to those of General Safety.
PLANTS FOLLOWING PRACTICES
PLANTS NEGLECTING SAFE PRACTICES
GENERAL SAFETY PROVISIONS
INDUSTRIAL EYESIGHT SUPERVISION
GOGGLES OR OTHER PERSONAL PROTECTIVE EQUIPMENT
* Snellen Chart examination omitted
** As determined by plant
Each symbol represents 10% of all plants surveyed
National Society for the Prevention of Blindness, Inc.
1790 Broadway, New York City

FIGURE II

*Reprinted from *The Sight-Saving Review*, Vol. XII, No. 4, December, 1942.

Expansion of industry and acceleration of production of war materials have placed a heavy responsibility on the eyes as well as on industry. Visual efficiency is of utmost importance in the war effort, both from the point of view of speeding up production and of avoiding accidents. In order to learn the extent of safe practices as they relate to eyesight in typical plants, the National Society for the Prevention of Blindness undertook a survey of industrial plants based on response to a questionnaire and interviews.

The slide now shown summarizes, in pictograph form, the results of this survey. The study covered 50 plants with a total of 166,862 workers. The plants which were surveyed employed from 12 to over 25,000 workers. Twenty-two plants had more than 1,500 employees, 10 plants had from 500 to 1,500 employees, and 18 less than 500. Small industries have not, as a rule, followed safe practices in their plants to the extent that such practices are followed in the larger plants. For this reason, small as well as large plants were included in the survey.

The limited budget available for this survey made it essential to plan the inquiry along lines which would give the most reliable information from the plants which could be visted or otherwise checked. Accordingly, the questionnaire will be found to contain items of general safety interest, as well as those directly relating to the protection and best utilization of eyesight in industry.

The study revealed that practically all plants carry out nearly all of the general safety procedures, such as providing first aid facilities (100 per cent); having definite safety programs and definite policies for the enforcement of safety rules (98 per cent); employing safety supervisors, full time or part time, according to the size of the plant (94 per cent). While the study took into consideration general safety procedures, it was mainly concerned with the protection of eyesight and the part played by good eyesight in speeding up production.

Under this heading are considered those factors having an immediate bearing upon production, reduction of spoilage, utilization of workers with subnormal vision and workers with "old sight." Despite the fact that front rank companies provide good general safety facilities, they, for the most part, appear to be unaware of the importance of eyesight in industry as a managerial responsibility.

Job analysis for visual requirements is claimed as a practice by 24 per cent of the plants. Since only 10 per cent have the cooperation of an ophthalmologist, we may consider that only these give adequate attention to this very important subject. Seventy-six per cent of the plants make no study to determine what visual requirements are necessary or acceptable to qualify a worker for any particular job. This means that they do not know how many color-blind or one-eyed men, or men with subnormal but correctible vision, can be utilized. On the other hand, they may be employing men whose vision is a hazard on the particular job. For example, a man may be working as a crane operator who has deficient "depth perception," and so cannot judge the height and placement of the crane load. This would make him a menace to life and property, while if assigned to another job for which his eyesight is suited, he could carry on safely and effectively.

Visual examination is made by eye physicians in 10 per cent of the plants; 20 per cent more employ binocular instruments; this leaves 35 plants — 70 per cent of the total — in which no test of employees' vision is made at all, or the test made is not adequate to determine the essential factors of industrial vision. For example, a Snellen chart alone will determine only the central vision of the worker for distant objects.

Periodic eye examination of workers who are especially exposed is reported by 14 per cent of the plants. Only 10 per cent of the plants report a general periodic examination of all workers. This is serious oversight because some industrial eye injuries are cumulative — for example, those due to intense radiation as in welding and furnace work.

Prescription lenses, in this discussion, refer to those goggles provided with nonshatterable lenses which have been ground to compensate for the visual defect of the employee. Prescription lenses are provided for, or required of, the workers in 58 per cent of the plants. This leaves 42 per cent in which no attention is paid to this subject. Only 34 per cent of the plants provide prescription lenses at company expense, and, in most of these, provision is made only for workers who are required to wear goggles. The

opportunity to raise productive efficiency of others who need eye correction is lost.

According to informed opinion, about one-fifth of general accidents result from bad "seeing" conditions, whether subnormal vision, deficient lighting, or glare. It might be expected, therefore, to find these items included in accident reports. However, only 22 per cent of the plants report the eye condition of the persons involved, and only 12 per cent mention the illumination at scene of the accident. The lack of these data suggests that a much greater percentage of general accidents may be caused by bad eyesight or poor illumination, or both, than is generally supposed.

The average plant investment per worker in machine industries is about $6,000. Most employers agree that, according to the nature of the work, from two weeks to two months is required before a new employee is capable of working at standard performance. Why then should there be any hesitancy in making an adequate examination at the cost of a few dollars to make sure that the new employee is being trained on that job at which he can work to the best advantage of all concerned?

Much more might be said of all of the items in this section, but the importance of eye conservation as a factor for maximum production of war materials may be epitomized by the statement that a substantial increase in production may be realized through an adequate program for eye conservation and illumination — this in addition to the humane results of preventing loss of sight or injury to the eyes.

According to the experience of Dr. Albert C. Snell of Rochester, New York, in his private practice, 90 per cent of eye accidents have occurred while goggles were not being used. These figures are in line with conservative estimates of other authorities. In fact, most authorities agree that at least 90 per cent of injuries to the eye can be completely prevented simply by wearing suitable protective equipment. In a very large majority of instances, such equipment is merely a pair of industrial safety glasses. Much complaint has been heard on the part of management that workers refuse to wear the goggles provided. Before we blame the men too much, let us look at the failure on the part of management in some of the front rank companies.

Eighty per cent of the plants claim to supply goggles to workers whom they consider "exposed." This figure is doubtless in excess of the fact, because some exposures are frequently not recognized. For example, some front rank plants do not consider that a lathe operator is exposed to eye injuries!

Only 70 per cent of the plants fit the equipment for the comfort of the worker, although safety men agree that this is essential to gain cooperation from the workers in the wearing of protective equipment. In 30 per cent of the plants, no attempt of any kind was made to fit the goggles or other equipment to the worker. That means that neither the plant doctor, the safety man, nor the foreman paid any attention to this feature.

In many plants, goggles are turned in and re-issued, and we find that more than a third failed to sterilize the goggles before issuing them to be worn by another workman. Does it not appear that these failures to follow the practices of common sense and ordinary hygiene may account, in large measure, for the difficulty in getting 100 per cent effectiveness in the "goggle" program?

TREATMENT OF INTRA-OCULAR FOREIGN BODIES

ALBERT C. SNELL, M.D.
Rochester, N. Y.

Before undertaking the treatment of eyes with intra-ocular foreign bodies it is obviously necessary to make a diagnosis of the presence of a foreign body within the globe. It is also necessary to determine the kind of matter of which the foreign substance is composed, whether magnetic or otherwise; the tissues through which the foreign body has passed, and its location within the globe.

Fragments of iron make up the major proporation of substances which become lodged within the globe. Other substances are glass, stone and wood. Ninety per cent of the metals consist of magnetic iron. Other metals may be copper, brass, aluminum, lead and non-magnetic steel.

A **diagnosis** of intra-ocular foreign body is usually not difficult when the injured eye is seen soon after injury and especially when there is a visible penetrating wound through the cornea, iris and lens; or when a perforating scleral wound is found, and

when there is a clear history of accident indicating the possible source of the foreign body. Many penetrating bodies are relatively small, causing very small wounds, and since many are not seen soon after injury, the diagnosis in such cases is more difficult.

The sources from which intra-ocular foreign bodies originate are either from the tools and machines being used or from the metal being worked. Most commonly the accident occurs while the workman is using hand tools, but it may occur while the employee is operating punch presses, shapers, lathes, drill presses, shears; in fact, foreign body injuries may occur from almost every type of metal cutting, trimming, boring and polishing machine from which particles of metal are thrown with force.

Most intra-ocular foreign bodies, because they are small, cause very little pain at the time of the injury — less than a foreign body on the cornea — so that the injured person often is not aware of any serious injury until later when either the eye becomes painful or the individual notices dimness of vision. Hence the patient often does not consult the ophthalmologist at the time of the injury. The history of the injury and the presence of one or more of the four cardinal signs will strongly indicate the probability of an intra-ocular foreign body. The four cardinal signs of recent perforating injuries are: (a) The presence of a perforating wound, (b) a soft eyeball, (c) a shallow anterior chamber, and (d) loss of vision. Anyone or all these cardinal signs may be absent especially when the injury is not seen soon after the accident. Later, one often must depend on the history alone in making a diagnosis. Penetrating wounds caused by small foreign bodies passing through the conjunctiva cannot always be discovered by inspection as these wounds often seem superficial and may heal over very promptly. Hence many of these cases when seen by an ophthalmologist, many days after the time of injury, are difficult to diagnose from the symptoms alone when most of the early signs are absent.

The history of an accident and the nature of work being performed often are the only leads to arouse one's suspicions of the probability of intra-ocular foreign body. When this is merely suspected, a roentgen examination is always indicated as the positive way to determine the presence or absence of an intra-ocular foreign body. When one feels reasonably sure of the diagnosis, the roentgen examination, careful localization and charting are essential. Whether the foreign body is lodged within or without the globe — that is, in the orbit — it can always be demonstrated in the x-ray film by the double exposure method. The extent that a foreign body may be imbedded in the scleral wall can not be determined from the roentgenograms.

Far too often has there been failure to diagnose the presence of intra-ocular foreign body until siderosis has taken place and vision lost. The industrial physician and (or) ophthalmologist must be alert to the possibility of this type of accident. Failure to make an early diagnosis has been responsibile for many blind eyes.

I shall discuss in this paper the treatment of cases in which foreign bodies lie posterior to the lens and its ligament. After determining the probability that the foreign body is magnetic, its removal by means of the electro-magnet must be attempted as soon as possible after the accident. However, a delay of not more than 24 hours does not make its removal more difficult. This delay may be necessary in order to obtain proper roentgenograms and essential localization charts. But when the delay is much longer, permitting encapsulation or the formation of inflammatory adhesions about the foreign body, it may become difficult or impossible to dislodge it. Encapsulation is most likely when the foreign body is lodged in the sclera, where most are found to be. Hence the indication for early operation. Foreign bodies which lie in the vitreous are not encapsulated so soon as those which are lodged in the sclera, and can frequently be removed quite easily even after months have intervened. In such cases even when the diagnosis and treatment have been delayed for several months, an attempt at removal of the foreign body is the proper practice as a few of these eyes can be saved.

Having charted the position of the foreign body posterior to the lens, one must then choose the type of magnet and the route by which to attempt extraction. The large hand-size magnet should be the one of first choice. The giant magnet is a second choice. It rarely succeeds, in the experience of the writer, when the hand magnet has failed, provided the proper technic has been used. The hand magnet will succeed in 98 per cent of the cases

where it is at all possible to accomplish extraction, whether the foreign body lies in or anterior to the lens, or posterior to it.

When the foreign body lies posterior to the lens and its suspensory ligament, the posterior or scleral route is the one to be chosen, since it is generally acknowledged to be the one by which the least damage is done in the process of extraction. When the lens remains clear and undamaged the anterior route is contra-indicated. The scleral route being chosen, the site for incision must be decided upon. This decision is based on two factors, "the zone of choice" and the position of the foreign body. "The zone of choice" is bounded by two concentric circles, 6 mm. and 10 mm. from the corneal limbus. These circles define a zone 4 mm. wide encircling the eyeball. An incision made in this zone does no damage to the ciliary body as this structure is anterior to the zone. Furthermore, the ora serrata lies within the zone, and it is the safest part of the eye to penetrate. The foreign body must lie nearest to some meridian on the globe (unless it should lie directly in the center of the eyeball). This meridian and the position of the foreign body is determined by data provided by the localization chart. The chart will also show how far the foreign body lies behind the lens, how far above or below a horizontal line and how far to nasal or to temporal side of a vertical line. These two lines cross at the center of the cornea. In selecting the site of the incision one should avoid the extra-ocular muscles; an incision to either side of a muscle does not place it materially farther from the foreign body. It may be noted that by thus selecting the site for the incision, it is not always made as near as possible to the charted position of the foreign body except when it lies in the zone of preference and not under any extra-ocular muscle.

Having selected the site for incision in the preferred zone and on the meridian indicated, a sufficiently large conjunctival flap including subconjunctival tissue is so made that the incision can be completely covered. The dissection is carried down to the sclera and all bleeding is checked. Two or three silk sutures are now placed through the edges of the conjunctival flap and placed loosely out of the field where the scleral incision is to be made. Then with a Graefe knife a crucial or T-shaped incision is made which may be enlarged if necessary with scissors. The knife or scissors should not enter the vitreous. The incision should be only slightly larger than the estimated or measured size of the foreign body.

The electro-magnet fitted with a cone-shaped blunt tip should first be used, placed in contact with the sclera over the incision before turning on the current. When current is turned on the metal usually is immediately attracted to the magnet and is easily extracted. If it is not, then the current should be snapped on and off several times. This procedure is often successful. But when it is not, one may place the tip directly over the localized area of the foreign body in contact with the sclera and with the current on, moving the tip toward the incision. Also one may try other shaped tips, some pointed, which may be introduced a millimeter or two into the vitreous through the incision.

Should one fail in all these attempts with the use of the hand magnet, the giant magnet should then be tried. As a last resort, enter one blade of a small pair of fine scissors into the incision while in contact with the magnet and direct the point towards the foreign body. Should all of these steps fail to remove the foreign body located posterior to the chosen zone, a second attempt may be made on the following day, through a new incision made as nearly over the foreign body as possible. First, try the hand magnet and then the giant magnet, using the same technic as described above.

Before closing the scleral incision the margins should be treated either by careful application of pure phenol as recommended by Stieren (Arch. of Ophth. 29:135, 1943), or a saturated solution of trichloroacetic acid, or by diathermy. Either of the above mentioned drugs is best applied by means of a sterile soft wooden tooth pick moistened in the solution. Avoiding any excess of fluid, one should carefully apply the tip to edges of the wound. If diathermy is chosen the round ball-like tip is recommended. By any of these methods the incision is adequately sterilized and a moderate plastic inflammatory reaction is produced which aids the closure of retinal and choroidal tears, thus tending to prevent detachment of the retina—a possible complication but a rare one when the foreign body is small and the above treatment is followed.

When detachment of retina is discovered early it is usually successfully reattached by the usual diathermy method of treatment. Very large foreign bodies are most likely to cause detachment of the retina and serious damage to other structures. Eyes thus damaged hold little chance for preservation of vision by any method of treatment including removal of the foreign body.

In all cases of intra-ocular foreign body the oral administration of a sulfa drug is recommended, to be continued from four to six days. The local after-treatment consists of instillation of atropine, an antiseptic ointment and a dressing.

Removal of Non-Magnetic Metal—Non-magnetic intra-ocular foreign bodies which lie in the vitreous in such a position that they can be seen with the ophthalmoscope can be removed when the lens is uninjured. Otherwise their removal is most difficult and generally impossible. If the foreign body can be seen with the ophthalmoscope, an incision should be made in the "zone of choice," described above; through this incision a special, small, fine forceps is entered with blades closed. The application of the forceps is then directed by observing with the ophthalmoscope its position, course and approach to the foreign body; then the blades of the forceps are opened sufficiently so that one may pass above and the other below the foreign body. After grasping it, it is easily removed. The scleral wound is treated and dressed as described after removal of magnetic intra-ocular foreign bodies.

Foreign bodies which have penetrated the eyeball but are lodged on the inside of the globe opposite to wound of entrance and posterior to the equator are most difficult to remove. In cases where the foreign body lies anterior to the equator and is definitely localized, one may succeed occasionally in its removal by cutting directly down on it. In case operation is likely to cause further and extensive mutilation of the eye expectant and conservative treatment is the best procedure.

Glass embedded in the sclera may remain indefinitely without causing change in vision. Other substances also may become encapsulated in the sclera without doing further harm than the original damage. Particles of iron are most dangerous due to the degenerating effect of siderosis, but even these when small and embedded in the sclera may be harmless. However, every reasonable attempt, which does not cause too great mutilation of the eye, should be made to remove iron intra-ocular foreign bodies.

SUMMARY

In the treatment of intra-ocular foreign bodies the important considerations are: (1) The determination of the presence of a foreign body; (2) Its definite location should be determined by roentgenograms and charted; (3) The probable kind of substance, especially whether or not magnetic.

Foreign bodies which lie posterior to the lens should be removed by the posterior route through an incision made in "the zone of choice."

The large hand magnet will succeed in 98 per cent of cases, but the giant magnet should be used when the smaller one fails.

The scleral incision should be treated with phenol or with trichloroacetic acid.

The removal of non-magnetic foreign bodies should be attempted when feasible by grasping the foreign body with suitable instruments.

It is good practice to administer orally a sulfonamide drug in all cases of perforating eye injury.

Dr. J. G. Townsend, Medical Director, Chief of the Division of Industrial Hygiene of the United States Public Health Service, was asked to state what the U.S.P.H.S. found to be the most crucial problems of Industrial Ophthalmology. The following is his reply:

Problems in industrial ophthalmology as lined up by the U.S.P.H.S., Washington, D. C., fall into four general categories:

1. The ordinary diseases of the eye, found in industrial workers which probably would be the same as in the general population.
2. Industrial accidents affecting the eye which, of course, would apply only to the group of industrial workers and would not be found to such an extent in the general population.
3. Industrial diseases, such as optic neuritis due to certain toxic exposures and epidemic keratoconjunctivitis, which seems to affect workers, probably due to group contact, but which also can appear in communities outside the plant.

4. Visual defects which need refraction, especially on precision jobs. Here again, the same ration of visual impairment would be found as in the general population, except in those industries where eyestrain or precision jobs would aggravate the existing condition if not corrected.

Taking these up in sequence, we have a few figures on conditions under category number 1. Dr. Gafafer has analyzed reports on a group of approximately 3,000 male employees of an industrial plant. During a five-year period, the 3,000 employees in this plant experienced 100 eye disabilities (accidents excluded) causing absence from work of one day or longer. The 100 disabilities were distributed as follows, according to diagnoses made by physicians and nurses:

Sore eye	25
Conjunctivitis	15
Eye operation (including cyst and cataract)	8
Ulcer	7
Iritis	7
Stye	6
Eye infection	6
Cold in eye	3
Uveitis	3
Eyestrain	2
Pink eye	2
Dacryocystitis	2
Miscellaneous	14

During this five-year period, these 3,000 male workers were recorded as having 11,830 one-day or longer absences (all causes, excluding accidents), 100 of these absences were attributable to eye disabilities. Moreover, the 11,830 absences accounted for 97,559 days of disability, the eyes accounting for 1,195 days. Thus, the eye accounted for 0.85 per cent of the absences, and 1.22 per cent of the days absent. This does not represent a large absentee total for ordinary ocular diseases, but from the nature of the diagnoses, such as "sore eye" and "cold in eye," it would seem that certainly a consulting ophthalmologist should be available to plants, as the physicians on duty are not trained, naturally, in the finer technics of ophthalmological diagnoses.

Our second category of industrial accidents does not give us, according to our records, any definite figures as to the number of such accidents occurring in a given group, but this figure can be obtained, as you well know, from the National Safety Council in Chicago. These figures show, for example, that 4 per cent of all industrial accidents are eye injuries and these injuries represent 5 per cent of all compensation costs for injuries. Of course, the correction of this condition could be simple, in the wearing of goggles and corrected vision goggles where defects of vision do occur. In our general inspection work in ordnance plants, we find this practice generally adopted, although it does depend so much on the cooperation of management and labor to insist that goggles be worn in the trades where the eye is in danger. It should be recognized that very many interruptions in the day's work, amounting to hours in many cases and often most of the working day, result from foreign bodies in the eye. Most of these are not recorded. This group may very well represent a greater loss in man-hours than from reported eye injuries.

The solution of the problem of the third type of industrial eye injuries—namely, industrial diseases, per se — I suppose would have to be treated as cases occur. A very definite statement has been prepared, as you know, for the prevention and treatment of epidemic keratoconjunctivitis, which has been very widely circularized, and I think most industrial physicians are aware of what to do.

I think that one of the most important problems, and probably the most difficult in solution, is the proper refraction of the eyes of workers needing such treatment, which is the fourth group mentioned in this letter. I do not know that in the individual worker's file, either in the medical department or the personnel department of the plant, there is a record of visual acuity. By and large throughout the nation, this particular defect has not been tabulated to give a very clear picture as to just what is the percentage of refractive errors among workers. In our Manual of Industrial Hygiene, on page 61, there is mentioned your survey of 16,332 industrial employees of several plants, in which was found that acuity defects of 20/40 or less ranged from 15 to 38 per cent, and that muscle imbalance, sufficient to interfere with comfortable and efficient vision, averaged 25 per cent. About 20 per cent of the applicants had visual defects that were sufficiently high to handicap their efficiency. It is altogether reasonable to assume that similar surveys would give approximate figures. I think

it is highly important, especially in precision jobs, that such corrections be made and that glasses be furnished at a nominal cost.

In our Manual, on page 62, there is the following statement, on which I would appreciate criticism from the Committee:

"Role of Optometrists. — In some war industries optometrists visit the plant and make refractions, especially where corrections are provided for safety glasses or goggles. Because of the shortage of physicians on account of the war, it will probably become necessary to bring optometrists into the picture to a greater extent. To insure satisfactory work and to prevent misunderstandings, joint committees of plant physicians, ophthalmologists and optometrists should be formed to consider the roles of each of these groups in sight conservation."

In your letter you ask a direct question as to what to do in the case of an ordnance plant 40 or 50 miles outside of town, in which there might be neither an ophthalmologist nor an optometrist. You continue, "Such an ordnance plant might require refractions and corrected vision goggles. What is the answer?" I do not believe I can answer that categorically, except that I question whether one would find any ordnance plant 40 or 50 miles distant from an ophthalmologist or an optometrist. However, if so, the only solution that I see is for the plant to make some arrangement with the nearest ophthalmologist or optometrist, or both, to visit the plant on a consultant basis to make such corrections as may be necessary and to prescribe the glasses. Of course, in some plants will be found physicians who are capable of making refractions. Quite frankly, I do not know the index of this accomplishment among the industrial physicians located in very many plants throughout the country. I might suggest that the Committee furnish state industrial hygiene divisions a list of available ophthalmologists and optometrists who could be called in by the various plants for this service.

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Committee Activities

COMMITTEE ON STANDARDIZATION OF TONOMETERS

Report on Tonometer Checking Stations

When Schiotz devised his useful instrument he established a manufacturing shop in Oslo to produce instruments according to his specifications which were sold under his certification. A reference standard instrument of this manufacture was carefully calibrated by Schiotz in a series of investigations extending over more than two decades. On the basis of these calibrations, Schiotz drew up his chart, with the aid of which tonometer scale measurements can be interpreted in terms of millimeters of mercury intra-ocular pressure.

While there has been throughout the years some discussion as to the reliability of Schiotz's calibration, there is general agreement that the measurements which he made constitute a close approximation to the truth. In order that the Schiotz scale should have this degree of validity, however, it is necessary that it should be used with an instrument which conforms accurately to the specifications originally made by Schiotz himself.

Unfortunately in the course of years a great many manufacturers in Europe, also in this country, have undertaken to construct Schiotz tonometers without any supervision or control by the profession. As a result, instruments have been placed on the market which deviate more and more from the original Schiotz instrument and from one another. The extent of these deviations has gained momentum in recent years to a degree which is really shocking. Thus, for instance, an otherwise reliable American instrument manufacturer has been producing a tonometer in which the plunger, when resting on the eye with its usual load, weighs between 6½ and 7 grms. though it is still labeled 5½ grms. As a result, errors in measurement of as much as 30 and 40 per cent are common, and wide discrepancies in clinical evaluation occur, particularly in borderline cases where accurate tonometric measurements are essential.

This unhappy state of affairs led the American Academy of Ophthalmology and Otolaryngology, at its meeting in 1942, to establish a Committee on Standardization of Tonometers. The present note represents the first report on the activities of this Committee.

Even before this Committee had been established, a very important step in the same direction had already been undertaken by Doctors Schoenberg and Posner under the auspices of the National Society for the Prevention of Blindness. With the aid of funds from this Society, a tonometer checking laboratory was established by Doctors Schoenberg and Posner in the central offices of the National Society for the Prevention of Blindness in New York, and methods were devised in this laboratory for accurately measuring the physical characteristics of tonometers sent them for checking. The reports of their preliminary findings have been published.* These reports show that only a small percentage of the tonometers which they examined correspond even reasonably well to the specifications laid down by Schiotz.

Following the important lead given in this field by the National Society for the Prevention of Blindness, the Committee on Standardization of Tonometers has established two other checking stations, one in Chicago under Dr. Peter Kronfeld and another in San Francisco under Dr. Frederick Cordes, and the work of these three checking stations has been closely correlated. Tonometers sent to any of the three stations are being similarly measured.

Some of the tonometers submitted to the checking stations have been found to be true Schiotz Tonometers, and instruments reported as being satisfactory by any one of the three stations can be expected to read similarly on the same patient.

Many of the instruments tested are found to differ significantly from the standard Schiotz instrument. Doctor Kronfeld has worked out approximate correction factors for the Schiotz chart to be applied to the most common errors found in Schiotz instrument construction, and those instruments which are otherwise mechanically satisfactory are being returned to their owners with a corrected Schiotz chart applicable to the individual instrument for which the corrections have been calculated. When the tension is measured by such an instrument, with the aid of its own corrected calibrating chart, the results are found to be in good agreement with those obtained with a standard Schiotz instrument.

A considerable number of the instruments submitted to the checking stations are found on examination to have minor mechanical defects which can be corrected by minor repairs. Both in New York and in Chicago, mechanics have been found who are able to make such repairs if the owner of the tonometer so desires. After the repairs have been made the instrument is re-examined in the checking station and returned to its owner with a final report.

Finally, a considerable number of the instruments submitted to the checking stations are found to be so defective that no corrections and repairs can be made to put them into reliable working condition. Such instruments are returned to their owners with the advice that they be discarded.

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* A Checking Station for Tonometers. Arch. of Ophth. V. 14, p. 453, Sept., 1935. By Adolph Posner.

Standardization and Checking of Schiotz Tonometers. Arch. of Ophth. V. 3, pp. 1-30, July, 1943. By Adolph Posner.

The Technique of Tonometry and Care of Tonometers Followed at the Glaucoma Clinic of the Manhattan Eye & Ear Hospital of New York. By Mark J. Schoenberg. In press.

A report on Defects Found in Tonometers Examined at the Checking Station of the National Society for the Prevention of Blindness. By Mark J. Schoenberg and Adolph Posner. In press.

The work of all three checking stations is supervised by the Committee on Standardization of Tonometers and an annual report of the work of the stations will be rendered to the Academy. A charge of $2.50 is to be made for each instrument tested in the hope that this work may become self-supporting.

The Committee does not claim that by the work of these checking stations, the reading of tonometers can be made absolutely accurate and reliable, for much depends on how the tonometer is used by the individual physician, but it is confidently expected that the chief sources of discrepancies between measurements with different Schiotz Tonometers will be greatly reduced. Furthermore, the Committee is endeavoring to induce American manufacturers of Schiotz Tonometers to agree on standard specifications so that the vagaries in the construction of these instruments can be eliminated. The results of the work of the Committee in this field will be reported separately.

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PAPER CONSERVATION VITAL TO ALL

Unless American industry reduces its tonnage consumption of printing paper sharply during the first three months of 1944, there is grave danger that certain types of printing will have to be restricted or eliminated altogether. This information comes from the Joint Committee on Government Relations of the Commercial Printing Industry of the U. S., which, with the support of the WPB, is conducting a vigorous printing paper conservation program.

Already Canada has sharply restricted the "end-use" of printing paper and certain types of advertising have been eliminated entirely; most printing is subject to licensing restrictions and before printing jobs can be ordered an application must be made to a rationing board. For the first quarter of 1944 the printing industry of the United States has been allocated 75 per cent of the paper tonnage used during the comparable period in 1941.

In order to comply with the request of the WPB to save paper the supplements to the TRANSACTIONS and reprints of scientific articles will be produced with self covers only.

W. L. BENEDICT, *Managing Editor*

AMERICAN ASSOCIATION OF EYE, EAR, NOSE AND THROAT SOCIETY SECRETARIES

W. Howard Morrison, M.D., *President*
Kenneth L. Craft, M.D., *Secretary-Treasurer*

The fifth annual meeting of the American Association of Eye, Ear, Nose and Throat Society Secretaries was held Monday, October 11, 1943, at the Palmer House, Chicago, during the convention of the American Academy of Ophthalmology and Otolaryngology. The meeting was held at a luncheon provided through the courtesy of the Academy, which has sponsored this secretarial group since its organization.

Dr. W. Howard Morrison of Omaha presided at the meeting which was attended by 35 secretaries of various E. E. N. & T. societies from all parts of the United States. The program was in the able hands of Dr. William E. Keith, secretary of the Kansas City Society of Ophthalmology and Otolaryngology and director of the Seventh District of this society. Doctor Keith introduced the following topics for consideration and general discussion:

1. "To Fee or Not to Fee"; the question of the proper financial relationship between the doctor at home and his colleague in military service whose former patients are being cared for by the stay-at-home.
2. A plan for establishing a central clearing house for scientific articles, case reports, movies, etc., for the benefit of E. E. N. & T. societies and the American Academy of Ophthalmology and Otolaryngology.
3. Post-war status of E. E. N. & T. societies.

A full report of the annual business meeting of the officers and directors of this association will be published in the next issue of the Transactions.

There was a free discussion of these subjects by various members as follows:

To Fee or Not to Fee. Dr. Sheldon B. Freeman, Buffalo, New York, Secretary of the Buffalo Ophthalmologic Club:

"In asking me to speak a few words on this topic, President Morrison no doubt refers to the subject which I brought up at the meeting last year concerning members of our local societies who are not able to carry on a private practice because of the fact that they are serving in our country's military forces. Perhaps you will recall that last year I said the Buffalo Ophthalmologic Club had passed an amendment to the Constitution which makes provisions for a member called to active, full-time military duty. First such a member is exempt from paying dues, and, second, whenever another member of the Club takes care of a patient who would have been taken care of by the absentee doctor if he were not in the military service, one-half of the fee collected shall be turned over to the doctor in military service. Further, the doctor who is away is kept informed of the local society's activities by the Secretary so that the home contact is not entirely lost.

"As far as I know, this plan has worked out in a very satisfactory manner. The opticians do not have any knowledge of the plan and do not cooperate in any way. Whatever is done is a responsibility of only the local doctor. I have kept records of money received, and when a sufficient amount has accumulated to make a sizeable sum, a

check has been sent to the doctor in service. The only complication that I have had is that patients frequently state that their own doctor took care of them at a special reduced rate, and I have found it necessary to take the patient's word for this and collect the fee charged by the regular doctor, according to the patient's statement.

"I believe that this plan can be recommended for any local society."

Dr. Thomas F. Furlong, Jr., Philadelphia, Secretary of the Philadelphia Laryngological Society:

"All the members of our society and also of our section, taking care of any patient of a doctor from the Philadelphia area who has gone into service, sees to it that 50 per cent of such fee or fees collected from said patients are sent to the doctor on active duty, to his attorney or to his wife, if he is on foreign service. Records of such disbursements are necessary for clarification of one's income tax. From personal experience and the experiences of other members of our society and the section, this has entailed merely a small amount of extra secretarial work, and we feel it is the least we can do for our fellow members who are doing their bit."

This subject was also discussed by Dr. W. A. Ohmart, Denver, Colorado, Secretary of the Colorado Ophthalmological Society, and by Dr. Harold F. Whalman, Los Angeles, Secretary of the Research Study Club.

A Plan for Establishing a Central Clearing House for Scientific Articles, etc. Dr. Thomas F. Furlong, Jr., Philadelphia, Secretary of the Philadelphia Laryngological Society:

"Regarding Doctor Keith's suggestion for the establishment of a central clearing house for papers, reports, movies, etc., for exchange with other societies and the Academy, I heartily agree to this. However, may I make the additional suggestion that one of the paid secretaries in Doctor Benedict's office be made responsible for this chore. Most of us here hold office for a short period of time, and I have noticed that since 1905, a period of 38 years, the Academy has had only five secretaries. Hence, I feel that the main office of the Academy should handle this matter."

Dr. Paul C. Craig, Reading, Pennsylvania, Secretary of the Reading E. E. N. & T. Society:

"This plan is excellent. It opens a new field of endeavor for the practicing eye, ear, nose and throat physician and perhaps for the Academy. It will provide a broader base of teaching and understanding, and it will give the man from the small town opportunities for increased participation in the activities of the Academy. The Reading Eye, Ear, Nose and Throat Society is wholeheartedly in favor of this suggestion."

Dr. Sam H. Sanders, Memphis, Tennessee, Secretary of the Memphis Society of Ophthalmology and Oto-Laryngology:

"Doctor Keith's suggestion for establishing a central clearing house for any information of importance to the Association is a very good one. I am sure we could all benefit by such a procedure, but details would have to be worked out. A full time secretary would be essential for the work."

Dr. Gabriel Tucker, Philadelphia, Pennsylvania, College of Physicians:

"This is my first meeting with this group, and I have enjoyed it very much. I am sure that this group will do a great deal to help improve and maintain the high character of the programs in the various societies and close cooperation among the members of the various

groups. I believe that this work you are doing is most important for the perpetuation and the improvement of various similar societies. You and your associates are to be complimented on the work that you are doing.

"I know of the activities of Dr. Paul Craig of Reading and also of Dr. Thomas Furlong of Philadelphia, and I am sure that their work is an inspiration to us all for the preparation of better organized programs. I want to thank this society for the privilege of meeting with you."

This matter was also discussed by Dr. Harold F. Whalman, Los Angeles, Secretary of the Research Study Club.

Post-War Status of E. E. N. & T. Societies. In the discussion of the post-war status of E. E. N. & T. societies, the point was made that a number of these societies has disbanded due to loss of membership to the armed forces and lack of interest in the face of the war situation. The general feeling was that this attitude emphatically should be discouraged and that these tottering societies should be urged and encouraged to continue active organization throughout the duration. These thoughts led rapidly to a general discussion of socialized medicine by Dr. James J. Monahan, Shenandoah, Pennsylvania, Secretary of the Lower Anthracite E. E. N. & T. Society, and Dr. Edley H. Jones, Vicksburg, Mississippi, Secretary of the Louisiana-Mississippi Ophthalmological and Oto-Laryngological Society. Action taken upon this matter will be transmitted directly to all secretaries by the directors of the various districts.

The meeting was adjourned by Dr. W. Howard Morrison.

KENNETH L. CRAFT, M.D., *Secretary*
1002 Hume Mansur Building
Indianapolis 4, Indiana

Those present were:

Dr. John H. Matheson, Iowa Academy of Ophthalmology and Otolaryngology, Des Moines, Ia.

Dr. Kenneth L. Craft, Indianapolis Ophthalmological and Oto-Laryngological Society, Indianapolis, Ind.

Dr. Glen G. Gibson, Philadelphia County Medical Society, Philadelphia, Pa.

Dr. Walter H. Theobald, Chicago Laryngological Society, Chicago, Ill.

Dr. Burton E. Lovesey, New England Oto-Laryngological Society, Boston, Mass.

Dr. F. D. Hurd, Montana Academy of Ophthalmology and Oto-Laryngology, Great Falls, Mont.

Dr. Sheldon B. Freeman, Buffalo Ophthalmologic Club, Buffalo, N. Y.

Dr. E. M. Keown, Colorado Oto-Laryngological Society, Denver, Colo.

Dr. J. Kent Leasure, Indiana Academy of Ophthalmology and Oto-Laryngology, Indianapolis, Ind.

Dr. W. A. Ohmart, Colorado Ophthalmological Society, Denver, Colo.

Dr. Arthur Hammond, Detroit Oto-Laryngological Society, Detroit, Mich.

Dr. Harold F. Whalman, Research Study Club, Los Angeles, Calif.

Dr. Orris E. Ghrist, Los Angeles County Ophthalmological and Oto-Laryngological Society, Glendale, Calif.

Dr. George McGeary, Minnesota Academy of Ophthalmology and Oto-Laryngology, Minneapolis, Minn.

Dr. Alton V. Hallum, Atlanta E.E.N. & T. Society, Atlanta, Ga.

Dr. Verne Lance, Georgia State Medical Society, E.E.N. & T. Section, Atlanta, Ga.

Dr. W. A. Mann, Chicago Ophthalmological Society, Chicago, Ill.

Dr. Edley H. Jones, Louisiana-Mississippi Ophthalmological and Oto-Laryngological Society, Vicksburg, Miss.

Dr. Sam H. Sanders, Memphis Society of Ophthalmology and Oto-Laryngology and Tennessee Academy, Memphis, Tenn.

Dr. Forney P. George, Central Pennsylvania E.E.N.T. Association, Harrisburg, Pa.

Dr. Paul C. Craig, Reading E.E.N. & T. Society, Reading, Pa.

Dr. James J. Monahan, Lower Anthracite E.E.N.T. Society, Shenandoah, Pa.

Dr. John B. McMurray, Pittsburgh Ophthalmological Society, Pittsburgh, Pa.

Dr. Daniel S. DeStio, Pittsburgh Otological Society, Pittsburgh, Pa.

Dr. J. McClure Tyson, Western Pennsylvania E.E.N.T. Society, Dubois, Pa.

Dr. Archie E. Cruthirds, South Western Academy of E.E.N.T., Phoenix, Ariz.

Dr. Charles T. Sullivan, Eye, Ear, Nose and Throat Club, Rochester, N. Y.

Dr. A. A. Levin, Cincinnati General Hospital, Cincinnati, Ohio

Dr. W. E. Keith, Kansas City Society of O.O.R.L., Kansas City, Mo.

Dr. W. H. Morrison, Omaha and Council Bluffs E.E.N.T. Society, Omaha, Nebr.

Dr. T. F. Furlong, Jr., Philadelphia Laryngological Society, Philadelphia, Pa.

Dr. J. C. Decker, Sioux Valley Eye and Ear Academy, Sioux City, Iowa

Dr. Gabriel Tucker, College of Physicians, Philadelphia, Pa.

Dr. R. H. Criswell, Saginaw Valley Academy of Ophthalmology and Oto-Laryngology, Bay City, Mich.

Dr. Lester A. Brown, Shreveport E.E.N.T. Society, Shreveport, La.

Pan-American Congress of Otolaryngology

(Report of Committee on Inter-American Relations in Otolaryngology)

INTRODUCTION

One of the reasons for the establishment of the Pan-American Congress of Ophthalmology, according to Doctor Gradle, was the disruption of the International Congresses of Ophthalmology which had met every fourth year from 1857 to 1914, when World War I interrupted the series. These congresses were resumed only after a period of 15 years, with a meeting in Amsterdam in 1929. Subsequent meetings took place in Madrid in '33 and in Cairo in '37. Vienna was to have been the site of the next meeting, in 1941, but it could, of course, not be held because of the outbreak of World War II.

I should like to point out that an exactly parallel condition exists in the field of otolaryngology. The First International Otological Congress took place in the United States, under the presidency of Dr. H. John Roosa nearly 70 years ago, the next was held in Milan, and subsequent congresses met at Bale, Brussels, Florence, London, Bordeaux and Budapest; the ninth, the last before the outbreak of World War I, was held in Boston in 1912, with Dr. Clarence Blake as president. The war caused a hiatus of 10 years, but the Tenth International Congress of Otology was held in Paris in 1922 under the presidency of Professor Sebileau. This congress was not truly "international," because representatives of Germany and Austria were not invited. Six years later a Congress was held in Copenhagen under the presidency of Professor Schmiegelow, at which it was decided to give it the name First International Congress of Oto-Rhino - Laryngology instead of the Eleventh International Otological Congress. This Congress was a successor to the 10 Otological Congresses and three Laryngo-Rhinological Congresses, the last of which had been held in Berlin in 1911. It seemed as if the succession of International congresses was to be permanently resumed. A fine Congress of Oto-Rhino-Laryngology (the second) was held in Madrid in 1932, with Professor Tapia as president, and the Third International Congress of Oto-Rhino-Laryngology was held in Berlin in 1936 under the presidency of Professor Carl Von Eicken. This was a great success, and a well balanced representation of oto-rhino-laryngologists from many nations of the world participated. Arthur Proetz, Horace Newhart and I were among those who attended from the United States. The British, French and Belgian delegations were especially numerous, which seemed a sign pointing toward the development of real international friendship and collaboration. In Berlin it was voted that the next congress (the fourth) should take place in Amsterdam in 1940, under the presidency of Professor Burger. Plans for this congress were being energetically prosecuted until the spread of World War II made it obvious that cancellation would be necessary.

Thus, as in the field of ophthalmology, it seems appropriate that international cooperation in otolaryngology should be nurtured in the Western Hemisphere, where nations, though at war, are not at war with each other, and not yet become battlefields, even though their territorial waters be not immune from attack!

Now let us outline briefly what has already taken place in the direction of international cooperation in otolaryngology on the South American continent. In 1936 a meeting was held in Sao Paulo, Brazil, under the presidency of Dr. Mario Ottoni Rezende, which was called the Paulista Otorhinolaryngological Week. At this meeting it was decided to hold a First Brazilian Congress of Oto-Rhino-Laryngology, after an interval of two years. This First Brazilian Congress took place in 1938, in Rio de Janeiro, with Professor Marinho as president, and at its conclusion a decision was reached to join with colleagues of the other nations of South America in a First South American Congress of Otorhinolaryngology, this congress to be held in Buenos Aires with Professor Eliseo Segura as president. This First South American Congress of Otorhinolaryngology was held in 1940, and at its conclusion a first Latin American Congress was planned to be held in Sao Paulo, Brazil, in 1943, under the presi-

dency of Professor Paula Santos. Since it was deemed impractical to hold a Congress at this time, it was decided to hold instead, a Second South American Congress in Montevideo, with Professor Justo Alonso as president. In the light of this historical background, both in Europe and in South America, there can scarcely be any doubt of the advisability of taking steps toward the organization of a Pan-American Congress of Otolaryngology.

Organization of a Pan-American Congress of Otorhinolaryngology

Whenever and wherever the Pan-American Congress of Otorhinolaryngology meets, its problem of organization will be greatly simplified by the study of the excellent blue-print provided by the record of the Pan-American Congress of Ophthalmology as so ably set down in Doctor Gradle's report of May 1, 1943. I believe that the general plan of organization of the Pan-American Congress of Ophthalmology should be entirely applicable to a Pan-American Congress of Otolaryngology. The sending in invitations to all reputable otolaryngologists of the Western Hemisphere, as the ophthalmologists have done, would also be appropriate for the otolaryngologists. We have such a list in our possession. The papers presented at the Congress in English could equal the combined number presented in Spanish or Portuguese, using the same very efficient system of having translated abstracts thrown on the screen during the presentation of the papers. The use of a tri-lingual or quadri-lingual program, and the same system of registration, etc., which was used by the Congress of Ophthalmology could be adopted. A business meeting could be held at which officers such as those elected by the Pan-American Congress of Ophthalmology could be chosen: a President, several Vice-Presidents, two Executive-Secretaries and one Treasurer. The plan of establishing a Council in which each country should have one representative selected by the national society, or societies, or by the government of the particular country if there is no national society, seems excellent. Meetings should be held at two or three-year intervals, alternating between the Northern and Southern Hemispheres.

With regard to financing a Pan-American Congress of Otolaryngology, we may perhaps again profit by the example set by the ophthalmologists. According to Doctor Gradle's report, the American Optical Company, Bausch and Lomb Optical Company and Abbott Laboratories contributed a sum of $600.00 toward the expenses of their Congress. An effort could be made to solicit financial aid from similar organizations interested in otolaryngology: The companies dealing in hearing aid devices, drug companies and instrument manufacturers, for example. Among the drug companies, we already have some contacts with Sharpe and Dohme and with Smith, Kline and French. The subsequent financial maintenance of the Congress would doubtless depend on membership fees, registration fees and the rental of exhibit space at Congresses. Of course, it is to be hoped that the Academy may lend financial aid for the initial meeting.

Once the Pan-American Congress of Otolaryngology is organized, there will arise the necessity for the establishment of committees for the special study of various problems, for example the following:

Committee on the Prevention of Deafness

Committee on Allergy in Relation to the Nose and Throat

Committee on Specific Drugs in Otorhinolaryngology

Committee on Cancer of the Nose, Pharynx, Larynx, Bronchi and Esophagus

Committee on Peroral Endoscopy and Broncho-Esophagology

Committee on Professorial Exchange

Committee on Exchange of Graduate Students.

(The Kellogg Foundation sponsored 25 scholarships for graduate students in ophthalmology, and the selection of these students is being worked out with the cooperation of the Institute of International Education, a volunteer organization in New York City under the guidance of Dr. Stephen Duggan.)

History of the Committee on Inter-American Relations in Otorhinolaryngology

1942. On January 19, 1942, Dr. W. P. Wherry wrote me that the Program Committee of the Academy had appointed me as chairman of a committee to develop plans for a Pan American Congress of Otolaryngology, and that I was "given authority to appoint my own committee." He stated that the Program Committee

was desirous that "preliminary plans and a contemplated budget" be submitted to the Council at the Chicago Meeting of the Academy, October 11 to 14, 1942. Because of the increasing difficulties in communications and transportation resulting from our progressively more complete involvement in the war effort, nothing was done during the ensuing months except to write informally to some of my Latin American correspondents concerning the project of a Pan American Congress. I expected to be called upon for some sort of report at the 1942 meeting of the Academy, but after Doctor Wherry's death I hesitated to take the initiative in offering such a report. The annual meeting of the Academy came and went, and no report was asked for, but in December, 1942, Dr. W. L. Benedict was contacted and asked what action, if any, had been taken by the Council at the annual meeting.

I informed him that I had accumulated a considerable amount of useful information through correspondence with friends in the Latin American countries, which I should be glad to present briefly whenever and wherever was deemed appropriate. At Doctor Benedict's suggestion I got in touch with Dr. J. A. Babbitt, President of the Academy, whom I found deeply interested in the project of promoting closer inter-American relations in otolaryngology and establishing a Pan-American Congress.

1943. Throughout the spring months of 1943 Doctor Babbitt and I worked intensively. We sent letters to a number of representative otolaryngologists in the various other American republics, acquainting them with the fact that the Academy planned to have an Inter-American Night as a feature of the 1943 meeting, and asking them if they could accept an invitation to attend this affair, as well as participate in the scientific sessions of the Academy and to discuss with our Inter-American Relations Committee and the officers and Council of the Academy the project of establishing a Pan-American Congress of Otolaryngology. A preliminary report on these activities, and particularly the status of our correspondence with the Latin American colleagues, was presented at the special program-arrangements Committee meeting at the Waldorf-Astoria, New York, June 8, 1943.

Chicago, 1943. Despite the great difficulties in communication and transportation due to chaotic international conditions imposed by war, a very gratifying response rewarded our efforts. A number of distinguished Latin American otolaryngologists made the trip to Chicago; a number of others accepted the invitation to attend, but were prevented from reaching Chicago by circumstances beyond their control, and the great majority of those who did not accept wrote us expressing their appreciation of the invitation and their warm sympathy with the idea of establishing some sort of Pan American organization in otolaryngology.

Those who actually came to Chicago and participated in the seveal meetings of our Committee were, in the alphabetical order of the names of the countries they represented, the following:

Brazil — Professor Jose Kos of Rio de Janeiro

Cuba — Dr. Pedro Hernandez Gonzalo (President, Cuban Otolaryngological Society)
Dr. Abelardo Codinach

Haiti — Dr. Georges Hudicourt

Mexico — Dr. Ricardo Tapia Acuna

Salvador — Dr. Jose Guillen Rivas

Venezuela — Dr. L. Lopez Villoria
Dr. Victorino Marquez

An addendum at the end of this report summarizes the responses received from colleagues in these same and other countries.

The entire meeting of the Academy was pervaded with the spirit of inter-American friendship and cooperation. It was very appropriately keynoted by the magnificent address of the Guest of Honor, D. Ralph Fenton, on "The Unity of Medical Science in the Americas" on Sunday afternoon. At the Inter-American Relations Dinner on Wednesday night President Babbitt welcomed the Latin American guests in Spanish, and they were then introduced individually. Addresses of the evening were given by Dr. William L. Schurz, of the Division of Cultural Relations of the State Department and Colonel Albert Dreisbach, of the Office of the Coordinator of Inter-Ameican Affairs.

Our Committee met for the first time at breakfast on Sunday morning, October 10. President Babbitt greeted the members of the Committee and the Latin

American guests, and then Doctor Gradle was asked to speak with regard to the project of a Pan American Congress of Otolaryngology from his experience in connection with the establishment of the Pan American Congress of Ophthalmology. He outlined briefly the story of the ophthalmological organization and made numerous suggestions regarding the proposed otolaryngological one. He stated that the Second Pan American Congress of Ophthalmology, postponed from 1943, had been tentatively planned to be held in Montevideo in 1944.

Various members of the Committee and the Latin American colleagues present discussed the project of a Pan American Congress of Otolaryngology. A number of suggestions were made as to the best place to hold a First Congress. Mexico City, Havana, New Orleans, New York and Montevideo were among the cities mentioned. Several of the members of our Committee expressed the opinion that there would be numerous advantages in holding the first Pan American Congress in conjunction with, or immediately following, an annual meeting of the American Academy of Ophthalmology and Otolaryngology, as the First Pan-Ameican Congress of Ophthalmology was held. Subsequent congresses would, of course, be held in the other American Republics, perhaps in conjunction with the meeting of one of the Latin American societies.

Dr. A. W. Proetz pointed out that once plans were formulated and the site of the Congress decided upon proper publicity should be obtained in all the otolaryngological publications of the Americas.

Dr. Paul H. Holinger stated that he believed one of the most important functions of a Pan American organization would be to facilitate the exchange of postgraduate students and residents. He suggested that, even now, our Committee might be able to accomplish something along these lines.

At the conclusion of the meeting it was moved by Dr. Ralph A. Fenton that THE ACADEMY BE REQUESTED TO SPONSOR THE ESTABLISHMENT OF A PAN AMERICAN CONGRESS OF OTOLARYNGOLOGY AND TO FINANCE ITS INITIAL MEETING, THIS INITIAL MEETING TO BE HELD IN CONJUNCTION WITH A MEETING OF THE ACADEMY. This motion was seconded by Dr. Walter H. Theobald, and passed.

At a second meeting held Monday, October 11, at 5:00 p.m., Dr. Juan H. Font, of Puerto Rico, spoke in both English and in Spanish, giving some helpful suggestions, and several members of the Committee also spoke. Various matters pertaining to the promotion of inter-American relations and the establishment of a Pan-American Congress were further discussed.

At a third and final meeting, held Wednesday afternoon in Doctor Babbitt's suite, Doctor Babbitt thanked the members of the Committee and the Latin American guests for their cooperation in laying the ground-work for a Pan-American Congress during his administration, and promised that he would continue to do all he could to further the project.

Plans and Recommendations for 1944

The Committee will continue as both a Committee on Inter-American Relations in Otolaryngology and a Committee on Pan American Congress of Otolaryngology, with the same make-up as last year, except that Dr. Victor Alfaro is to be added, and that Doctor Babbitt is now to be a regular member of the Executive group, instead of an ex-officio member of the Committee; and further, that Dr. Gordon B. New is asked to serve as a member of the Executive group, and that Doctor Post is asked to serve ex-officio as President. The following is a complete list of the Committee as it now stands:

Executive Committee

Chevalier L. Jackson, Chairman
James A. Babbitt
Ralph Fenton
Juan Font
Arthur Proetz
Frank Spencer
Gordon B. New

Members

Victor Alfaro
Howard C. Ballenger
Wesley Bowers
Mackenzie Brown
Thomas Carmody
George M. Coates
William Gill
Perry Goldsmith
Paul Holinger
Charles J. Imperatori
Francis Lejeune
James M. Robb

Wm. E. Sauer
Leroy Schall
Marshall Taylor
Walter Theobald

Consultants

Harris P. Mosher
Moacyr Alvaro
Conrad Berens
Harry Gradle
Forrest J. Pinkerton

Ex-Officio

Lawrence T. Post, President
Wm. L. Benedict, Executive Secretary

Our plans and recommendations for 1944 are as follows:

(1) We plan to keep in touch with all the Latin American colleagues who attended the 1943 meeting, and invite them to return in 1944. We shall also keep in touch with the other Latin American colleagues with whom contact has been established, and establish contact with additional ones.

(2) Letters will be written to the various societies of otolaryngology in the other American republics, acquainting them with the interest of the American Academy of Ophthalmology and Otolaryngology in the project of a Pan-American Congress and extending to them an invitation to join the Academy in organizing such a Congress.

(3) A special letter is being prepared to be sent to Professor Justo Alonso, President of the Second South American Congress, acquainting him with the deliberations of our Committee and the action of the Academy in granting the request of our Committee that the Academy sponsor the establishment of a Pan-American Congress of Otolaryngology and finance its initial meeting, to be held in conjunction with an annual meeting of the Academy, probably in 1945. Professor Alonso has already indicated to us his approval of the idea of organizing a Pan-American Congress, and he has expressed his willingness to invite as delegates to his Second South American Congress members of the Academy whose names our Committee sends him. It is suggested that interested members give their names to the Chairman or to some member of the Committee.

(4) Our Committee will undertake to formulate a definite plan for a first Pan-American Congress to take place in conjunction with the annual meeting of the Academy in 1945. In formulating this plan we shall hope to have the collaboration of a number of our Latin American colleagues, and when the plan is completed and has been approved by the Council of the Academy, it is suggested that it be proposed to the President and Executive Committee of the South American Congress in the form of an invitation to join with the Academy in organizing the first Pan-American Congress.

(5) Preliminary notice of the projected Pan American Congress of Otolaryngology sponsored by the American Academy of Ophthalmology and Otolaryngology will be sent to all the specialty journals of the Americas, the text of this notice to be approved by the Executive Secretary of the Academy.

(6) Preliminary steps will be taken in carrying out the suggestion of Doctor Holinger that our Committee endeavor to "promote the exchange of postgraduate students and residents in otolaryngology between the United States and the other American Republics." A form letter is being prepared to be sent to all the institutions offering approved residencies in otolaryngology, asking them what opportunities they have to offer for Latin American postgraduate students and residents.

(7) A definite time should be set aside on the 1944 program of the Academy for a meeting of our Committee possibly at a breakfast or lunch, on the first day of the meeting. If possible, a special meeting of the Committee will be arranged at the time of the meeting of the American Medical Association in Chicago.

ADDENDUM

The following is a summary of the responses received from otolaryngologists of the other American republics to unofficial letters asking their views regarding the project of a Pan-American Congress of Otolaryngology and inviting them to attend the 1943 meeting of the Academy.

ARGENTINA:

Professor Eliseo Segura, Dean of South American Otolaryngologists and President of the First South American Congress of Otolaryngology (1940) replied promptly to our letter, offering his enthusiastic sup-

port in the establishment of a Pan-American Congress of Otolaryngology, but regretted that he would not be able to come to Chicago in October, 1943.

Dr. Santiago L. Arauz acknowledged our letters and stated that he would have much pleasure in accepting the invitation but at present could not definitely promise to come. He stated that since he was proposed for the Chair of Otolaryngology he would have to remain in Buenos Aires.

Dr. Roger Lanza Castelli (Cordoba) was very anxious indeed to attend the Chicago meetings and he had been named as the official representative of the University of Cordoba, but he was unable to obtain priorities for transportation.

Dr. Abelardo Irigoyen Freyre (Santa Fe) cabled that the impossibility of obtaining priorities would prevent his attendance at the meetings but that he was "spiritually present" and that furthermore he would promise his collaboration in the organization of a Pan-American Congress.

Dr. Manuel Gonzalez Loza (Rosario) replied to our invitation that he would like very much indeed to be able to accept but that he could not, in view of the present difficulties, see his way clear to planning the trip. Furthermore, he expressed his opinion that there would be no chance of realizing a Pan-American Congress of Otolaryngology until after the conclusion of the war.

Among the other Argentinian colleagues to whom letter were written, but who either failed to receive them or could not accept the invitation to come to Chicago under present conditions were:

Dr. Juan M. Tato (Buenos Aires)
Dr. Antonio Zambrini (Buenos Aires)
Dr. Richardo Bisi (Buenos Aires)
Dr. Jose Manuel Aliaga (Cordoba)

BRAZIL:

Professor Jose Kos (Rio) attended the meetings in Chicago making a special trip for the purpose.

Dr. Plinio Barretto (Sao Paulo) cabled expressing his interest and offering full cooperation but stating that he would not be able to come to Chicago. (Dr. Barretto has since been in this country and has offered many helpful suggestions. Furthermore, he will be in Rio, Montevideo and Buenos Aires in the very near future, and he will personally convey to the otolaryngologists of those centers, and particularly to Professor Alonso, a message from our Committee.)

Among the other Brazilian colleagues whom we have contacted or are attempting to contact are the following:

Prof. Joao Marinho (Rio)
Dr. Aristides Monteiro (Rio)
Dr. Raul David de Sanson (Rio)
Dr. Elmiro de Lima (Rio)
Prof. Paula Santos (Sao Paulo)
Dr. Mangabeira Albernaz (Sao Paulo)
Dr. Mario Ottoni Rezende (Sao Paulo)
Dr. Arthur de Sa (Recife)

These men either did not receive our letters or were unable to consider coming to Chicago for the past meeting. However, it is believed that most if not all of them would be favorable to our project.

CHILE:

Dr. Anibal Grez had accepted our invitation and made all plans to come when the grave illness of his father prevented him from leaving.

Dr. Juan Barroilhet sent a most informative and helpful letter in which he stated that he could not himself come to Chicago but that he was heartily in favor of the project of a Pan-American Congress.

Dr. Alfedo Alcaino stated that he could not attend the meeting in Chicago because of "reasons of war."

COLOMBIA:

Dr. Cleves Vargas regretted that he could not, in the time allowed him, complete arrangements to make the trip.

CUBA:

Dr. Pedro Hernandez Gonzalo, President of the Cuban Society of Oto-Laryngology, attended the Chicago meetings, making a special trip for the purpose.

Dr. Abelardo Codinach likewise made a special trip from Havana to attend the Chicago meetings, representing the Cuban Society of Otolaryngology and also the City of Havana.

DOMINICAN REPUBLIC:

Dr. Manuel Pastoriza replied enthusiastically to our invitation and stated that he would be glad to attend the meetings if the Dominican Embassy in Washington could permit and direct him to do so. The time was not sufficient to arrange for such an appointment.

HAITI:

Dr. Georges Hudicourt, who is doing post-graduate work in this country, represented Haiti at the meetings with our Committee.

Dr. Elie Vilard, already a member of the Pan-American Congress of Ophthalmology, wrote enthusiastically about the project and promised his support.

MEXICO:

Dr. Ricardo Tapia Acuna attended the meetings in Chicago and offered many helpful suggestions.

Dr. Pastor B. Molina tried to attend but was unable to do so. He will plan to attend subsequent meetings.

(The Mexican Society of Ophthalmology and Otolaryngology will, I am quite sure, officially support a Pan-American Congress.)

PANAMA:

Dr. Sergio Gonzalez, head of the Department of Otolaryngology at the Santo Tomas Hospital, stated that he would be glad to come to Chicago if the government would send him as an official delegate. He promised to make every effort to attend the meetings next year.

PARAGUAY:

Professor Crispin Insaurralde (Asuncion) acknowledged our invitation, stating that his attendance at the meeting this year would be entirely impossible, but he spoke most enthusiastically about the project of a Pan-American Congress.

PERU:

Dr. Alberto Flores, distinguished Peruvian otolaryngologist, and President of the Peruvian Academy of Surgery, deplored the fact that he could not accept our invitation, because he was obliged to attend the Second Inter-American Congress of Surgeons in Buenos Aires, scheduled for the same dates.

Dr. Ernesto Raffo acknowledged our invitation, stating that he would be delighted to attend the meeting of the Academy if it were not for the "present abnormal circumstances."

PUERTO RICO:

Dr. Juan Font, a loyal member of the Academy and a member of our Committee, attended the Chicago meetings and expressed his views fluently in both English and Spanish. He promised his full cooperation.

EL SALVADOR:

Dr. Joaquin Guillen Rivas came to Chicago, but unfortunately arrived too late to participate in the meetings of cur Committee.

URUGUAY:

Professor Justo Alonso, President of the Second South American Congress of Otolaryngology, which is to take place in Montevideo this year, replied most enthusiastically to our invitation, regretting his inability to attend the meetings in Chicago, and stating that he, as well as the Society of Otorhinolaryngology of Uruguay, was disposed to collaborate in the idea of the realization of periodical Pan-American Congresses of Otorhinolaryngology. Professor Alonso offered to invite whatever colleagues from the United States we believed might be able to attend the Second South American Congress scheduled to meet this year in Montevideo under his presidency.

Dr. Felix Leborgne, distinguished radiologist and author of an excellent book entitled "Cancer of the Larynx, a Radiologic Study," was very anxious to accept our invitation, and had all plans made to come to Chicago, when unexpected circumstances of a personal nature prevented him from leaving.

VENEZUELA:

Dr. L. Lopez Villoria attended the Chicago meetings and offered many helpful suggestions in connection with our plans for a Pan-American Congress of Otolaryngology. Dr. Lopez Villoria made the trip to Chicago especially to attend these meetings.

Dr. Victorino Marquez, who is studying in this country at the present time, also attended the meetings and participated in discussions. (Dr. Marquez has been of very great help to the Committee in translating correspondence into Spanish.)

Dr. Franz Conde Jahn acknowledged our invitation but regretted the impossibility of his attendance this year.

Dr. Julio Garcia Alvarez likewise acknowledged our invitation and spoke enthusiastically about the idea of a Pan-American Congress but was unable to arrange to make the trip this year because of reasons beyond his control.

Contacts are now being established with otolaryngologists in Bolivia, Costa Rica, Ecuador, Guatemala, Honduras and Nicaragua, and with additional colleagues in the countries in which we have already established contact.

CHEVALIER L. JACKSON
Chairman

In Memoriam

George Robert Gowen.....Walla Walla, Washington
September 2, 1943

Joseph Edward Miller.....Los Angeles, California
September 13, 1943

Otis F. Simonds.....Cleveland, Ohio
September 26, 1943

George Foster Fiske.....Chicago, Illinois
October 18, 1943

Zenas Horace Ellis.....New York, New York
October 20, 1943

Louis Sam Smith.....Brooklyn, New York
October 23, 1943

William Alfred Shoemaker.....St. Louis, Missouri
November 7, 1943

Max Bakst .....Brooklyn, New York
November 10, 1943

Arthur E. Gadbois.....Norfolk, Nebraska
November 19, 1943

W. F. Clevenger.....Indianapolis, Indiana
November 20, 1943

Max Lubman .....New York, New York
November 20, 1943

Charles Curtis Jones.....Cincinnati, Ohio
November 22, 1943

Herbert Maskell Goddard.....Philadelphia, Pa.
November 23, 1943

Charles H. Brobst.....Peoria, Ill.
November 25, 1943

Edmond E. Blaauw .....Buffalo, N. Y.
December 4, 1943

Charles Henry May .....New York, N. Y.
December 7, 1943

Raymond Duane Sleight.....Battle Creek, Mich.
December 7, 1943

Carl F. Schaub.....Chicago, Ill.
January 7, 1944

Myron Metzenbaum .....Cleveland, Ohio
January 25, 1944

Frank L. Secoy .....Sioux City, Iowa
January, 1944

SURGERY OF THE LABYRINTH FOR MÉNIÈRE'S DISEASE

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THE TITLE of this paper may be ill-advised since there is considerable disagreement as to the meaning of the term "Ménière's Disease." As originally used it referred to a spontaneous hemorrhage into the labyrinth based on a case described by Ménière in 1861. The terms "Ménière's Syndrome" or "Ménière's Symptom Complex" were later used to designate the group of symptoms originally described by Ménière as being associated with his case but which were found to occur in many other conditions. In recent years the term "Ménière's Disease" has been more loosely applied to include all cases having the symptom complex and negative otoscopic findings. Dandy[2] has used the term of "pseudo-Ménière's" in cases in which only the vestibular part of the labyrinth was involved. We know that the Ménière's symptom complex may originate from a number of causes but little effort seems to have been made to subdivide the cases showing these symptoms into different classes. This group of symptoms consists of paroxysmal attacks of labyrinthine vertigo with or without nausea and vomiting, and usually associated with severe tinnitus and some degree of deafness. The symptoms may be of toxic origin and I have personally experienced them from food poisoning. They may be caused by allergens and a considerable number of cases have been reported with a proven allergic basis. They frequently occur as local manifestations of systemic conditions, such as syphilis and arteriosclerosis of the brain. The above groups often show a hyperirritable labyrinth. I believe that the term "Ménière's disease" should be limited to the group of cases which show symptoms and signs of distention or hydrops of the membranous labyrinth of unknown cause, the so-called idiopathic type. This is apparently a disease entity though the etiology and nature of it are still unsolved. It has been suspected for years that the cause of many of these recurring paroxysmal labyrinthine attacks is an increased intralabyrinthine pressure or glaucoma of the labyrinth. Whether this be due to an increased secretion of endolymph, a decreased resorption or a chemical imbalance has yet to be proven, but there is a sound basis for each of the three theories. Sufficient autopsy material has now been studied to show that hydrops or distention of the labyrinth does definitely occur and there are some dozen cases reported to date.[1]

I have been studying this subject for some years and believe I have seen enough cases to enable me to describe the symptoms and signs which should occur in such cases and which I have encountered in the group of cases I am reporting. They are as follows: intermittent paroxysmal attacks of vertigo with deafness and tinnitus of varying intensity and frequency often associated with nausea and vomiting and with negative otoscopic findings. In the early stages of the disease it is not uncommon for the vestibular labyrinth or the cochlea to be involved separately but, sooner or later, both parts of the labyrinth become involved. The deafness often varies in severity from time to time and usually involves the entire scale and is

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associated with decreased bone conduction, diplacusis and distortion. Tones are heard at a lower pitch in the affected ear. If the condition continues over a period of years, the deafness becomes more profound and shows little or no variation. The diplacusis and distortion are no longer noticeable because of the severity of the deafness. Audiograms suggest a uniform nerve deafness. Tinnitus is an almost constant accompaniment of the deafness and is usually of a double nature, a high-pitched ringing or hissing which is not bothersome and a low-pitched pounding or roaring usually synchronized with the heart beat which is most troublesome and which in some cases is complained of as bitterly as the vertigo. Caloric tests show either a nonfunctioning or hypo-active labyrinth on the affected side, and the reaction to the caloric tests may vary at different times. As a general rule, the more severe the deafness the less the labyrinth will react to the caloric test. A curious finding in many cases is the diminished reaction to caloric stimulation of the unaffected labyrinth which is especially true of the vertical canals. This may be due to nature's effort to balance or equalize the activity of the opposing labyrinths. The above symptoms and signs are suggestive of a varying endolymphatic distention with an accompanying variation in the sensitivity of the end organs involved. The acute attacks may be brought on in one of two opposite ways. First, an increase of pressure in one labyrinth may suppress its activity and may result in a physiological over-reaction from the opposing labyrinth. Secondly, the attack could be due to a temporary release or lowering of the pressure in the distended labyrinth so that this labyrinth "kicks in" to upset the balance control of the other one. A significant point is that during an attack the hearing in the affected ear may markedly improve. Between the acute attacks there is usually some deafness, the low-pitched tinnitus continues and the offending labyrinth is hypo-active to stimulation. Patients frequently complain of a constant sense of imbalance or unsteadiness between the active attacks and of a feeling of pressure or fullness in the head.

I am making no attempt to explain the underlying cause of hydrops of the labyrinth in this paper nor am I directly concerned with the consideration of the medical treatment, although in passing I might say that most of the forms of treatment advocated at present would seem to be definitely aimed at the distended labyrinth. These forms of treatment include the use of histamine, nicotinic acid, elimination or replacement of sodium, and dehydrating the patient. It has been my experience that conservative therapy seldom cures, although it often alleviates the symptoms and enables the patient to tolerate the condition. I do believe that conservative therapy should be tried. There are many cases which do not respond to conservative therapy and the condition is so incapacitating that the patients become desperate and are willing to gamble on any kind of surgical procedure which might eliminate this distressing malady. Moreover, the economic phase deserves consideration for many of these patients are unable to work or earn a livelihood and protracted medical treatment is out of the question for economic reasons.

The accepted surgical procedure at present is section of the vestibular nerve[2] and this procedure has been quite successful in a few expert hands. However, it is a hazardous undertaking and the results sometimes have been disappointing. The grinding or roaring tinnitus is usually not alleviated and some cases have cerebellar disturbances and difficulty in balancing, even though the acute vertiginous attacks disappear. Partial or total nerve section would

seem to be an illogical procedure for this condition, as it does nothing towards attacking the seat of the trouble, namely, hydrops of the labyrinth. I believe that any surgery performed should be upon the labyrinth itself and should include opening the membranous labyrinth to release some of the endolymph.

There have been few reports of labyrinth surgery for this condition. Portmann's[4] endolymphatic sac operation has relieved some patients although the relief has often been temporary. In England, the operation of choice seems to be alcohol injections into the labyrinth as reported by Wright[6] and Mollison,[3] either through the drum and oval window or through a trephine opening in the horizontal canal. This procedure causes complete destruction of the cochlea and vestibule and not infrequently has caused facial paralysis. It has found little favor in this country. Putnam[5] has reported 2 cases in which he opened the superior semicircular canal by way of the middle cranial fossa and used a coagulating current to destroy the vestibule without destroying the cochlea. His procedure seems to me to have considerable merit except for the fact that it is an operation by way of the back door.

My procedure has been as follows: A partial simple mastoidectomy is performed by the postauricular route, in which the antrum is opened widely and the outer wall of the aditus is removed far enough forward to expose the short process of the incus. With a small motor-driven burr, the horizontal canal is opened medial to the short process. The opening is made in this portion of the horizontal canal since it is near the ampulla and, through an opening at this point, a needle can be passed forwards through the canal into the vestibule. When the point of the needle strikes the anterior wall of the vestibule it is then slightly withdrawn and pressed towards the medial wall so as to avoid the facial nerve. A light coagulating current is then used. I have been giving two to three "shots" of about one second each. A few sulfa crystals are then dusted into the cavity and the mastoid incision is completely closed with clips. The clips are removed on the fourth day and the dressing usually discarded by the sixth day.

I have carried out this operative procedure on 8 cases suffering from Ménière's symptom complex whose symptoms and signs suggested a hydrops of the labyrinth. Six of these cases had unilateral involvement and 2 had bilateral involvement. The elapsed time since operation in 6 of the cases has been from one to three years and I believe this is sufficient time to enable me to make some evaluation of the results, although I know that some patients have spontaneous remissions lasting for months or even years. In all cases to date, the above procedure has resulted in a vestibular mechanism in the operated ear that is completely inactive to caloric stimulation. The hearing has varied tremendously. In 2 cases, there was total loss of hearing. In 2 cases, the postoperative hearing has remained at essentially the same level as before operation. Three cases showed further loss of hearing following the operation, affecting primarily the lower tones. In 1 case which had a uniform preoperative loss of hearing of about 70 decibels, the hearing has returned almost to normal since operation and has continued so for a year and a half. The 6 cases with unilateral involvement have been free of vertiginous attacks since the operation. Some had almost immediate relief from vertigo while others took from two to three months before the other labyrinth completely adjusted itself to the new situation. The amount of postoperative vertigo and imbalance seemed to be directly proportional to the labyrin-

thine activity in the affected ear as shown by caloric tests before operation. These 6 cases have been rehabilitated and have returned to their normal way of life and all have been most grateful for the operation. Moreover, the low-pitched grinding or pounding tinnitus has been completely relieved, although there has been little effect on the high-pitched tinnitus which in some cases increased. In none of these cases has there been any complaint about the residual high-pitched tinnitus. Two patients state that the pounding tinnitus before operation was as bad as the vertigo. In the 2 bilateral cases a good result was not attained. Unfortunately, these were the 2 cases in which complete loss of hearing in the operated ear resulted. It was hoped to preserve enough hearing in the operated ear to warrant a later operation on the other ear without endangering the ability to hear. Fortunately 1 of these cases responded later to histamine therapy and has been rehabilitated at least for the time being. It is curious to note that before operation this patient had an extensive course of histamine therapy without relief. There were no postoperative complications in any of the cases in this series. The wounds all healed by primary intention. The patients were well enough to be discharged from the hospital in from one to three weeks, depending upon the amount of postoperative physiological labyrinthine overaction from the unaffected ear. Incidentally, no gross distention of the membranous horizontal canal was noticed in any of these cases at operation.

I believe some comment on the effect of using a coagulating current in the vestibule in this operation is called for, although I can only conjecture as to what actually happens. The purpose of using the coagulating current is to destroy the vestibular mechanism with as little damage to the cochlea as possible. The coagulating current may result in actual coagulation of a part of the membranous organ so that the cochlea is sealed off from the vestibule, and the endolymph in the former is not lost. This would account for continued cochlear function following the operation. A needle passed through the horizontal canal into the vestibule would lie above the utricle and would perforate only the horizontal canal and its ampulla, unless the utricle were so greatly distended as to fill all available space. I do not know if the current destroys or coagulates the utricle and perhaps the saccule and thus destroys the vestibular function. It may be that the current destroys the fibers of the branches of the vestibular nerve to the utricle and superior semicircular canal which lie in the immediate vicinity of the end of the needle. It is hoped that we may be able to get sections of some of these labyrinths for pathological study at some future time. I can offer an explanation for the complete loss of cochlear function in 2 of the operated cases. I think this was owing to the use of the coagulating current when there was too little fluid inside the bony labyrinth resulting in a complete loss of endolymph. In these 2 cases, the labyrinth appeared to be empty after coagulation. It was not until I operated on the second case that this idea occurred to me. It had been my practice to flush out with saline the bone dust and debris from the mastoid cavity and then remove it with suction. I believe in these 2 cases the suction tip was allowed to come too close to the opening in the canal and resulted in the removal of the perilymph.

This operative procedure is not perfected and must be considered as still in the experimental stage. Nevertheless, I think it deserving of a report at this time since it has proven so successful in accomplishing its primary purpose, that of relieving the vertiginous attacks

of Ménière's disease. It is hoped that with modification there can be better assurance of the preservation of hearing. I am offering this in the hope that others may try my procedure and help attain this goal. Animal experimentation and histologic examination of labyrinths on which the procedure has been done are particularly desirable.

CONCLUSIONS

1. The operative procedure presented is still in its experimental stage.
2. The results in the series of cases presented seem to justify it as a surgical procedure for intractable cases of hydrops of the labyrinth.
3. It has proven successful in terminating the vertiginous attacks in such cases.
4. The effect on hearing has varied tremendously from restoration of normal hearing to a complete loss of hearing.
5. The low-pitched pounding or grinding tinnitus has been eliminated by this procedure although high-pitched tinnitus has sometimes increased.
6. This procedure seems to be preferable in its results, simplicity and safety to that of sectioning the vestibular nerve.
7. The procedure seems justified only in cases of labyrinthine distention or hydrops which fail to respond to medical therapy, or where complete destruction of labyrinthine function is desired.

CASE REPORTS

Case 1. S. L. The patient was a coal miner 29 years of age who had suffered for seven years from attacks of vertigo with low-toned tinnitus in the right ear. The attacks had been increasing in frequency and severity. Deafness was not noticed. He was incapacitated for three months previous to the examination with almost daily attacks of vertigo. There were frequent right sided headaches. He had been treated with a salt free diet in the hospital without effect. His general physical examination was negative. The examination for allergy was negative. There was slight deafness for low and high tones found in the right ear with decreased bone conduction. The Weber test was lateralized to the left ear. Caloric tests showed hypo-active response from the right ear, normal response from the left ear. November 12, 1940, a right labyrinthotomy with coagulation of the membranous canal was performed. A postoperative labyrinthine reaction existed for one week. There was instability on quick movements for six weeks. The patient went back to work after six weeks and worked steadily for the next nine months at which time contact with him was lost. Audiograms showed a uniform hearing loss throughout the scale averaging about 60 decibels. Tinnitus was not noticeable. No attacks of vertigo occurred after the operation. Result: Rehabilitation.

Case 2. M.W. The patient was a woman aged 31 who had a rough, low-pitched, pounding tinnitus in the right ear for three years previous to her examination. She had deafness and attacks of vertigo for two years prior to the examination. She was bed-ridden about half the time. Bilateral sphenoid operations had been performed. Treatments, including a salt free diet and histamine, were without effect on the symptoms. The response to allergy tests was negative. Examination showed a severe uniform deafness was present in the right ear with decreased bone conduction. Caloric tests showed hypo-active response in the left horizontal canal with no response from the right ear or left vertical canals. December 5, 1941, a right labyrinthotomy with coagulation of the membranous labyrinth was performed. There was slight labyrinthine imbalance following the operation. The patient was put on a full diet the next day. Eighteen months following the operation the patient was working in a defense plant. She had gained 30 pounds and had no vertigo since the operation. The pounding tinnitus was gone. Her hearing was practically unchanged. She had occasional slight high-pitched ringing noises in the operated ear. Result: Rehabilitation.

Case 3. M. O. R. The patient was a male, 55 years of age. He had attacks of vertigo associated with varying deafness in the left ear for eight years prior to examination. The onset of the attacks of vertigo occurred with a pounding roar in the left ear which was "worse than a machine shop." He had much treatment with drugs and diets. His last at-

tack occurred while driving a car and he crashed into a telephone pole.

The examination revealed a severe uniform deafness in the left ear which was present throughout the scale with decreased bone conduction. His blood pressure was 160/90. There was no response from the left vertical canals and a minimal response from the left horizontal canal on caloric stimulation. The right ear gave a hypo-active response from all the canals. March 6, 1942, a left labyrinthotomy was done with coagulation of the membranous canal. There was no postoperative dizziness. The patient sat up the next day and reported immediate relief from the loud tinnitus and marked improvement in hearing. Eighteen months after the operation his hearing was still good. There was no recurrence of vertigo. There was a slight occasional tinnitus like "frying bacon." He was able to work on a farm. Result: Rehabilitation.

Case 4. P. S. was a male patient aged 62. He had attacks of deafness in the right ear for thirty years and vertigo for twenty years before examination, with loud roaring in the right ear at the onset of attacks. There was residual hearing present in the right ear. The left ear became affected one and a half years before the examination, with increasing deafness and more frequent attacks of vertigo. The patient had much ineffective treatment. Caloric tests: There was a very slight response from the right ear and a subnormal response from the left ear. May 15, 1942, a right labyrinthotomy was done with coagulation of the membranous labyrinth. There was too much coagulation and the perilymph boiled away resulting in total deafness in the right ear. There was no postoperative vertigo and less tinnitus. The hearing in the left ear improved so that the patient could use a hearing aid with benefit. He was free from attacks for two months. Hearing in the left ear would be fairly good in the morning and gradually grow worse during the day. Two and one half months following the operation he had recurring attacks of vertigo from the left ear but not as severe as before the operation. Hearing would get better suddenly during the attacks and worse after them. Histamine therapy which was ineffective before the operation was again tried and proved effective in controlling the attacks and improving the hearing in the left ear. Result: Poor.

Case 5. F. C. I. The patient was a male 27 years of age. He had progressive deafness in the right ear for eight years and attacks of vertigo for two years preceeding the examination. Tinnitus was *not* a factor in his complaints. He received ineffective treatment with salt elimination, nicotinic acid and thiamin chloride. Caloric tests showed a minimal reaction in the right ear and decreased response from the left vertical canals. February 27, 1942, a right labyrinthotomy was performed with coagulation of the membranous labyrinth. There was no postoperative vertigo, recovery was uneventful and hearing was practically unchanged. One year following the operation the patient had gained weight, had no vertigo, and was working in a defense plant. Result: Rehabilitation.

Case 6. A. S. was a female aged 49. This patient had attacks of vertigo in the left ear for four years before examination. The attacks were associated with deafness and loud roaring tinnitus, with unsteadiness between attacks. She had ineffective treatment with diet, nicotinic acid and thiamin chloride. Caloric tests showed normal response from the right ear; hypo-active response from the left ear. August 27, 1942, a left labyrinthotomy was done with coagulation of the membranous labyrinth. There was moderate labyrinthine upset for one week following the operation and some unsteadiness remained for two months. She was unsteady in the dark for three months. In the following year there were no attacks of vertigo but there was a slight high-pitched hissing tinnitus. The pounding tinnitus was completely gone. The patient said that the tinnitus had been as bad as the vertigo before the operation. There was unnoticeable loss of hearing for low tones after the operation, the higher tones showing little change. Result: Rehabilitation.

Case 7. C. K. was a male patient 56 years of age. He had deafness and roaring in the right ear for seven years and attacks of vertigo for four years previous to the examination. The vertigo was somewhat relieved by treatment with nicotinic acid but there was no effect on the hearing or the tinnitus. During the two years before examination pounding in the left ear became worse than that in the right, accompanied with a varying, gradually increasing deafness. The patient was very sensitive to histamine and allergic to many tests but he failed to respond to histamine therapy, diet or the removal of offending allergens. The patient's father had committed suicide and the patient threatened to do likewise. One daughter of the patient also has Ménière's disease. Audiograms over a period of one year showed uniform deafness in the right ear with only moderate variations. The left ear showed wide variations from normal to a 50 decibel loss in the lower scale.

The upper scale showed a constant loss of about 50 decibels. During the six months before the examination the deafness of the left ear became more profound with fewer and shorter remissions of vertigo. There was no response from the vertical canals but a slight response from the horizontal canals of both ears on caloric stimulation. June 18, 1943, a left labyrinthotomy was done with coagulation of the membranous canal. The fluid in the vestibule boiled away resulting in a complete deafness in the left ear. A moderate labyrinthine upset occurred two weeks after the operation with subsequent unsteadiness for two months. There were several mild attacks of vertigo at intervals of one to two weeks. The patient was very despondent. Roaring in the left ear was better following the operation, but that in the right ear continued. The patient would not tolerate a hearing aid in the right ear, saying that there was too much distortion of the voice and that the sounds were too painful. Result: Bad.

Case 8. E. R. was a male, age 43. The patient had reduced hearing with throbbing low-pitched tinnitus in the left ear for about ten years previous to the examination. He had attacks of vertigo with nausea, vomiting and loss of consciousness for seven or eight years prior to the examination. The attacks became more frequent during the last two years. Unsteadiness and vertigo were present between the attacks. Treatment was ineffective. Caloric tests showed diminished response in both ears; the left ear was less active than the right. A moderate uniform hearing loss was present in the left ear with decreased bone conduction. June 21, 1943, a left labyrinthotomy was done with coagulation of the membranous labyrinth. A slight labyrinthine upset occurred for one week following the operation. There was some unsteadiness and imbalance for six weeks. Two months after the operation tinnitus was not noticeable. There were no dizzy attacks. The patient was back at work. His hearing was worse but not noticeably so to the patient. Result: Good to date.

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DISCUSSION

JOHN R. LINDSAY, M.D., Chicago, Ill.: Several months ago I had the opportunity of examining 4 of the patients operated on by Dr. Day. I can verify his statement that these patients reported complete relief from attacks of vertigo and that the hearing had been preserved in the operated ear.

Patients with severe Ménière's disease are primarily concerned with relief from vertigo and will willingly sacrifice hearing to obtain that result, but because the condition frequently becomes bilateral or occurs bilaterally, no treatment can be considered satisfactory unless preservation of the hearing can be assured.

Surgical treatment is reserved for cases which do not respond sufficiently to medical care. None of the medical treatments advocated has been completely satisfactory. A considerable number of these patients suffer severe disability unless they are given relief by surgery. Unfortunately, however, each of the surgical procedures used in the past had some undesirable feature.

The method reported by Dr. Day gives promise of overcoming most of these objectionable features in that the hearing has been preserved, the cranial cavity has not been opened, and the method is relatively simple and carries with it no danger to life. The question which remains to be determined is how much may be done in the way of surgical interference on the labyrinth without the probability of damage to the hearing, and also how much is necessary in order to eliminate the vertigo. I have carried out a number of operative procedures on the monkey and rabbit which I hope may help to answer these questions, when the work is finished. I have also operated on 3 humans, using a technic somewhat similar to Dr. Day's.

In 1 case fenestration of the horizontal canal was followed by insertion of a fine-

barbed dental broach into the ampulla in the same way that Dr. Day inserted the coagulation needle. Vertigo has been completely relieved in this case for over three years, but the hearing has been further depressed, although the labyrinth is not dead. A second case was fenestrated and coagulated in the ampulla, using a mild current. The patient has been free from attacks of vertigo for a year, but the hearing is profoundly impaired. In this case, may I say, fluid was boiled out of the canal when the coagulation current was turned on, leaving the canal empty at the end of the procedure. A third case had the horizontal and posterior canals fenestrated and the membranous canals destroyed. In this case also the extent of the surgical trauma was apparently too great, since hearing was profoundly impaired.

These patients, however, are all pleased because their vertigo has been completely relieved.

The question arises from Dr. Day's report as to whether the mild degree of coagulation used in his successful cases was really the important factor. Since the needle was immersed in fluid which was not allowed to boil away, it may be that no destruction of tissue took place within the labyrinth although the current was sufficient to stimulate the facial nerve. There seems a possibility that there may be no actual damage done in the vestibule, although, of course, the needle would probably penetrate the membranous ampulla. We cannot be sure as yet, but on the basis of our experience on the 3 humans and the experimental work done up to the preesnt, it appears very doubtful if any type of surgical trauma can be carried out in the vestibule without producing a reaction and fibrosis which would involve the auditory apparatus. A simple fenestration of one or more canals with a simple puncture of the membranous system may prove to be sufficient to eliminate the vertigo.

Dr. Day is to be congratulated on his excellent results. His report offers definite encouragement that this problem, which is primarily otologic, may be satisfactorily dealt with by relatively simple surgical procedure on the ear.

MARVIN F. JONES, M.D., New York, N. Y.: As time goes on I admire and envy the intestinal fortitude of the pioneer. This is a pioneer work, and when I think of putting a needle into the labyrinth and turning on a coagulating current, I have the same feeling that I have before I start to whistle going by a graveyard. There are methods of curing dizziness without access to surgery. Those methods have been outlined by myself in the few courses that I have given, and the important thing is to classify, as Dr. Day has suggested, the different causes and go after the cause in an intelligent fashion. There are about 35 known causes of dizziness.

In considering Ménière's disease or Ménière's syndrome, I think it is highly important that an exact definition be made of that term. It is being used so loosely that anything from the slight giddiness that one experiences in advancing years when arising in the morning; to actual prostration and dropping to the floor with a sudden loss of hearing and extreme tinnitus has been considered a Ménière's syndrome.

I have gone out on the limb so many times and found myself left there, in criticizing the work of others in the pioneer stage, that I am not going out on a limb in this instance. When any man with the honesty of Dr. Day does an experimental piece of work, I am going to hold my tongue very tightly until I have either tried it myself or someone that I know has tried it and substantiated or disproved the reported findings.

I have to congratulate Dr. Day on a very beautiful, fundamental piece of work, especially since he has courageously stated some of the symptoms that need explanation. One of the outstanding examples is the fact that a man can have an attack of dizziness and during that attack have an increase in his ability to hear. If we could only interpret and know what we hear and see we would go a great way toward establishing some of the fundamentals in our field that are so necessary before a real advance can take place.

CLARENCE SMITH, M.D., New York, N. Y.: In patients showing Ménière's symptom complex, there is an increase in the endolymph. Hallpike and Cairns,[1] in reporting the pathological findings in two undoubted cases of this kind, noted a gross distension of the endolymph system, together with degenerative changes in the sensory elements. This distension happens in certain pathological conditions. The increase in the pressure of the endolymph causes a lowering of the function of the affected ear. The inequality between the two ears causes the symptoms in Ménière's symptom complex.

In treating this condition surgically, in an effort to alleviate the symptoms, we have two main problems, one, to quiet the disturbed equilibratory function, the other, to preserve

the hearing. The question is, can we destroy part of the labyrinth and leave the remainder intact. We know that all parts of the membranous labyrinth communicate with each other and the contained endolymph is free to move from one position to another. We know that the ductus cochlearis commences in the lower part of the vestibule. Hallpike and Cairns found that the ductus cochlearis and the saccule are particularly prone to distension. They must be very near the ampulla of the horizontal canal. It would seem as though the coagulation, which evidently has to affect all the semicircular canals and the otolith membranes to completely destroy the vestibular function, might not stop there and could easily spread to the very near neighbor, the ductus cochlearis.

Dr. Day's records are infinitely better than those given for the Putnam[2] operation on the superior semicircular canals. He has apparently been brilliantly successful in destroying the vestibular portions of the labyrinths in his cases.

As a possible solution of the marked improvement in hearing postoperatively in the third case, I would suggest that this may have been brought about by the diminution of the patient's loud tinnitus. A subsidence of the tinnitus makes a marked difference sometimes in one's hearing ability. For instance, a patient of mine shows a loss of 53 per cent hearing in one ear when he is suffering from tinnitus, and only 28 per cent when that symptom is absent. The variation in hearing in Dr. Day's seventh case was possibly due to the same reason.

It would seem that the ideal case for this suggested operation would be one in which the hearing in the affected ear was bad.

Physiologically, destroying the labyrinth or cutting the fibers of the vestibular nerve coming from that labyrinth should yield the same results. The Dandy operation, when successful, spares the hearing while quieting the disturbed vestibular function. It is not, however, free from operative risk.

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H. M. Goodyear, M.D., Cincinnati, Ohio: Four years ago, on the theory that the symptoms of Ménière's disease were caused by hydrops of the membranous labyrinth, I did a decompression of the utricle by removing the bone between the ampullae of the horizontal and superior semicircular canals, extending the removal of bone as far anterior as possible. This opening of the bony labyrinth was covered by a thin skin flap from the posterior auditory canal, as I described in 1939 for the posterior approach in the fenestration operation. By the way, this fistula is still sensitive to the fistula test.

There were three severe attacks of Ménière's within ten days after operation, but in the four years following, only mild attacks of dizziness have occurred. However, it was evident that the operation was not a complete success and I made up my mind that I would remove or destroy the utricle in the next patient with Mèniére's symptoms who was available to me for operation.

I described the destruction of the utricle in 2 cases at our meeting here a year ago while conducting one of the instruction courses. The operation can be done under local anesthesia and, with deliberation, in less than an hour. My patients were able to walk on the fourth day and one left the hospital on the fifth and another on the sixth day after operation.

After the premedication and local injection, a posterior auricular incision is made. The cortex is opened with a trephine and burr, exposing the mastoid antrum and horizontal semicircular canal. The superior canal is outlined and an opening is made extending between the ampullae of the horizontal and superior semicircular canals by means of a small burr or a very small curette bent at forty-five degrees as designed by John D. Richards of New York. Once this bone is removed, sufficient space can be obtained for the passing of a small dental explorer which is passed to the inner wall of the utricle which is destroyed. The wound is then closed without drainage. Care is taken not to open or injure the posterior canal wall to avoid secondary infection from the external auditory canal.

In the 3 patients on whom I destroyed the utricle there have been no attacks of Mèniére's syndrome and each patient has been able to leave the hospital within a week after operation, getting about without support. The immediate reaction subsides rapidly. The first 2 cases were done more than a year ago; the third case was done more than four months ago. All these patients are well and very much pleased with the results of the operation.

In 1 of my cases hearing was retained for one week after the operation. To me it seems impossible to destroy the utricle without destroying the saccule, so intimately are they related and connected. It is difficult to explain how hearing remained in the cases reported by Dr. Day. It is possible in these cases that only the ampulla of the horizontal canal was destroyed by the coagulation as a measure of the destruction was impossible. That is, the needle was inserted and then withdrawn. We do not know just the distance it was withdrawn. Perhaps this limited destruction is all that is necessary to stop the symptoms of Mèniére's syndrome. Is it not possible that only the horizontal canal plays a part in these distressing symptoms?

We agree that in the presence of a cerebellopontine angle tumor there is no response from the homolateral canals while on the opposite side there is no response from the superior and posterior canals while the lateral canal still functions. Thus it is evident that the innervation of the horizontal canal is not the same as that of the superior and posterior canals.

Dr. McNally emphasized this morning that we have three semicircular canals but only two otolithic centers, one in the saccule which can be disregarded, and one in the utricle very near the mouths of the superior and horizontal canals, but chiefly at the mouth of the horizontal canal. Perhaps Dr. Day destroyed this otolithic membrane without destroying or draining the utricle in the cases with retained hearing.

Experimentally in the next case available to me for operation I shall determine this point by destroying only the ampulla or the immediate region of the horizontal canal, hoping to relieve the symptoms of Ménière's and at the same time retaining the hearing as occurred in the cases reported here.

KENNETH M. DAY, M.D., Pittsburgh, Pa.: I wish to thank my discussers for being so charitable. I expected them to wield the axe.

Dr. Jones spoke about getting out on a limb. I certainly felt that I was way out on a limb when I presented this paper to you today because there are so many unanswered questions about this procedure. It may be possible to puncture the horizontal membranous canal to release some endolymph and perhaps cure the dizziness and preserve the hearing. I don't know. I do know that the third patient who has normal hearing today, failed to respond to stimulation with ice water in the affected ear following operation. We have douched this man's ear on several occasions with ice water. He complains about it hurting but he does not have the slightest amount of vertigo. His vestibular labyrinth is apparently dead.

In these cases when we coagulate we get bubbling or boiling of the fluid and when I give the two or three coagulating shots, I watch with a magnifying glass to make sure the fluid bubbles. Remember that the facial nerve is very close. Already I should like to try modifying the procedure. However, I started on 1 case and this procedure worked. I did a couple more and it worked again and I am afraid to make any changes. My procedure was carried out on only a very few of over a hundred cases that I had under observation. The neurological and medical departments practically forced me to operate on a few of them. They seem to have more confidence in the procedure than I do.

This work certainly shows the great need of our going back to fundamental studies as Dr. Jones suggested. For instance: What about the endolymph, is it a real secretion or is it like the vitreous in the eye? Does the release of the endolymph relieve the disturbance? Does the release of too much endolymph destroy the hearing and does it not reform? We don't know. I think we have accepted too many dicta that have been passed on to us by our predecessors and the sooner we forget them and start again from scratch, the sooner we are going to begin learning something about the ear. I thank you.

ADVANCES IN TREATMENT OF CANCER OF THE LARYNX

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NEW YORK, NEW YORK

IN THE story of the fight against cancer, there is one bright page: the page that describes the control and cure of cancer of the larynx. On this page are italicized the events that emphasize the progress of this fight, beginning with the early attempts of Billroth in 1873 to remove the entire larynx and the tragic loss of his first 20 patients. The names of Sir St. Clair Thomson, Tapia, Solis-Cohen, MacKenty and Buckley stand out on this page as other milestones that mark the long and hazardous journey, from the early disheartening failures to what today we call almost complete success in the treatment of properly chosen cases of cancer of the larynx.

This story becomes an heroic epic when we read of the fight against this implacable foe: a struggle against infection, explosive and fatal hemorrhage, suffocation and pneumonia, esophageal fistula and the profound and hopeless mental depression that haunted the minds of the victims doomed to a lifetime without speech. These were problems that were solved by Sir St. Clair Thomson and MacKenty.

Sir St. Clair Thomson in a long and highly productive life brought the science of laryngology to the high state which it occupies today. His good results in treatment of cancer of the larynx by means of laryngofissure, a surgical procedure brought to perfection by him, gave us the first convincing proof that it was possible to cure cancer of the larynx.

This presentation will not include a report on my experience with the operation described as laryngofissure, the ideal operation for early cancer of the vocal cords. The majority of cases of cancer of the larynx which I have seen in private and hospital service have been so situated or so far advanced that total extirpation of the larynx was the treatment indicated.

To John E. MacKenty we owe a great debt for bringing to perfection the operation of laryngectomy. Through his efforts in the careful preoperative preparation of his patient, his skillful technic and his personal supervision of the postoperative care, Dr. MacKenty within a few years reduced the operative mortality of laryngectomy from over 50 per cent to less than 5 per cent.

CLASSIFICATION

All writers on cancer of the larynx have made use of a classification of this disease in an attempt to provide a universal language for the exchange of information. All have felt the need of some table or formula that would assemble, in an orderly manner, our accumulated knowledge of this subject so as to estimate the future course of the disease and thus aid our choice of treatment. The classifications offered have all been helpful and are used daily, with full recognition of their limitations. All classifications may be a source of danger if they are to be followed blindly as proven formulae.

The classification of cancers of the larynx on the basis of location, dividing tumors in this region into an extrinsic and intrinsic group, can be misleading because a tumor starting in the interior of the larynx may extend into areas that are extrinsic. An extension

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deep into the tissues may also exist that cannot be seen with the mirror or laryngoscope.

The laryngeal surgeon who has the best results as to cures and infrequent recurrences, will be the one who gives the correct value to the many factors that enter into the prognosis and choice of treatment of laryngeal cancer. This successful surgeon will know that the histological structure of a cancer is important. He will also know that the location, the tendency to invade surrounding tissue, the presence of local enlarged nodes and remote metastases, the physical condition and age of patient are all factors that do not admit of classification but do enter into our decision as to prognosis and choice of treatment. The experience and ability of the surgeon is a factor that must also be considered; a factor that will be difficult to classify.

PATHOLOGY

The histological examination of our material revealed that of 608 malignant epithelial new growths of the larynx, 603 were of the surface epithelial or epidermoid type of cancer. The squamous epithelium of the larynx may produce one of three types of malignancy: First, the basal cell type may be produced when the basement layer of cells alone is implicated. Second, the spinal or prickle cell type may be produced if these cells predominate in the growth. Third, when all three layers of the epithelium are involved and the squamous cells predominate with the formation of keratin and epithelial pearls, a tumor may be produced which is referred to as the squamous cell or epidermoid type of carcinoma. The great majority of malignant growths of the larynx are of the third or squamous cell type. This type of epithelioma is further divided into grades or types according to the classification of Broders. He grades malignancy according to the amount of anaplasia or the number of undifferentiated cells which tend to revert to the parent cell pattern. Broder's classification is roughly as follows: If about three-fourths of the tumor of a carcinoma is composed of differentiated and one-fourth of undifferentiated cells, it is grade 1. If the differentiated and undifferentiated cells are about equal in number, it is grade 2. If three-fourths of the cells are undifferentiated and one-fourth differentiated, it is grade 3. If there is such a lack of differentiation as to prevent identification of the adult type of structure, the tumor is grade 4.

A survey of our pathological material for the past 12 years reveals the incidence of epithelial malignancy of the larynx as follows:

Type 1	14
Type 2	526
Type 3	45
Type 4	11
Basal Cell	4
Spinal Cell	5

DIAGNOSIS

Repeated and careful mirror examination is of the utmost importance in diagnosis. The character of the growth, its location, the extent of involvement and degree of impairment of the mobility of the cord should be noted carefully. Fixation of a cord usually indicates a comparatively advanced growth.

A lateral roentgenogram of the neck as well as a planograph should be taken on all cases. Planography as brought out by Le Borgne a few years ago, is of considerable help in demonstrating the outline and extent of new growths in the larynx. A direct examination of the larynx should be done on all cases, the entire larynx and subglottic area should be inspected and a biopsy should be taken from the growth.

Too much emphasis cannot be laid on the importance of early diagnosis as a majority of the cases I have seen are

well advanced; often too far advanced for surgery.

It seems to hold true today as it did fifteen years ago when MacKenty stated, "Judging from the advanced condition of the great majority when they come for operation, the diagnosis of carcinoma of the larynx must be either extremely difficult or poorly understood by the profession. In hundreds of patients seen by me, a pathetically small number can be assured of a hopeful outcome. In many instances the fault lies with the patient in not seeking an opinion earlier but in far too many they have been under observation and treatment for months."

TREATMENT

The type of treatment chosen for cancer of the larynx should rest on the matured judgment of the surgeon. In making a decision as to choice of treatment, he should give proper value to the classification of the tumor, its size and location, the limitations of radiation therapy, and the age and physical condition of the patient. Credit without limit must go to the laryngologist who achieves a cure of a small epithelioma of the vocal cord by radiation therapy. His judgment in choice of treatment was correct. Nothing but adverse criticism is due the laryngologist who performs a laryngofissure, only to have a recurrence of the cancer in three months. His judgment and his choice of treatment were faulty.

Incipient cancer of the larynx seldom is seen in a hospital clinic. This type of case is suitable for the operation of laryngofissure and it is in this group of cases that 80 per cent of cures are obtained. It is my hope that as the result of education, many more cases will qualify for this operation within the next few years.

It is my opinion that the operation of laryngofissure should be reserved for those cases having a relatively small growth on one cord which does not extend across the mid line at the anterior commissure. I take this position with full knowledge of the success reported by other surgeons in obtaining good results by means of laryngofissure in cancers of the larynx that have spread across the anterior commissure and involve the opposite cord. During the past three years I performed laryngofissures on 3 such cases. The subperichondrial resection technic was followed. In all 3 cases, recurrence of the growth appeared within one year. Consequently, I have given up laryngofissure for growths extending across the anterior commissure.

ROENTGEN THERAPY

Roentgen therapy has shown great advances in the treatment of cancer of the larynx during the past ten years. In the early days of roentgen therapy it was not uncommon to see extensive burns and necrosis of the laryngeal cartilages from excessive dosage. But today with improved technic these complications are rare. Although better work is being done each year by radiotherapists I feel that roentgen therapy in laryngeal growths is still in the experimental stage and that surgery should be instituted wherever possible, followed by roentgen therapy if necessary.

The decision to follow the procedure of total laryngectomy is predicated on a number of factors. Due consideration must be given to the age and physical condition of the patient, the grade or type of cancer cell, the presence or absence of enlarged cervical nodes and to the extension of the growth to structures not amenable to surgical extirpation. When enlarged cervical nodes are present the larynx should be removed, followed by removal of the nodes by

dissection. This should be followed by irradiation of the cervical region.

LARYNGECTOMY

When the decision has been made that a total extirpation of the larynx is indicated, the patient is carefully prepared for several days preceding the operation. A detailed survey is made of the patient including blood serology, blood chemistry, and an estimation of his ability to accept sulfa therapy. A preliminary transfusion of whole blood has been made use of in certain cases with great benefit to the patient. Two days before the operation he is given 50 cc. of 5 per cent sodium sulfadiazine in sterile salt solution intravenously and the same dose repeated one day before. Since we have been using the sulfonamides in large doses—keeping the concentration in the blood around 10 to 12 milligrams per 100 cc.—the neck infections in these patients have been cut down considerably. Now it is not uncommon to see the esophagus heal without any fistula, and it is possible to remove the feeding tube from the fourth to tenth postoperative day. Used locally these drugs do not seem to influence the infection. Several days are set aside for instructing and training the patient in the use of the esophageal voice. This ingenious method of instruction before operation was suggested to us by McCall of Cleveland. One element that contributes in a large measure to our success in laryngeal surgery has been the training and employment of a surgical team. This team is made up of a surgical associate, a scrub nurse, two house officers and a supervising nurse who is a frequent observer in the operating room. She has been trained in the use of feeding tubes, aspiration of the airways, and the use of oxygen therapy.

The surgeon and his associates alternate in leading the team and so have equal opportunity to perfect themselves in technic. The team operates together daily and the postoperative care is continued by the entire group. This sort of work may be best described as work done by a number of associates, all subordinating personal prominence to the efficiency of the whole.

It was formerly our custom to get the maximum amount of extension of the neck to facilitate the dissection as soon as the patient was put on the table. However, in many cases, especially where the growth was large with little air space, patients had difficulty in breathing and it was necessary to open the trachea hurriedly. Of late the head is not extended until after the trachea is exposed and made ready for opening.

Local anesthesia is used. From 5 to 8 ounces of one-half of one per cent novocain with 15 minimums of adrenalin in the first ounce are used to infiltrate the entire neck from the symphysis mandibulae to the sternal notch and laterally well beyond the sternomastoid muscles. Careful attention is given to the deep injections about the hyoid, and on either side of the larynx and trachea. The operator and assistant inject at the same time, one taking one side of the neck and the other the opposite. In this way, the time for inducing anesthesia is cut down about ten minutes. The average time required to obtain anesthesia with two persons injecting is about five minutes.

A straight midline incision is made from just under the symphysis mandibulae to the sternal notch. The cricoid cartilage and isthmus of the thyroid are located at once. The thyroid isthmus is separated from the trachea, severed, clamped and sutured. This first step is taken so that the trachea may be opened leisurely at any later stage of the operation in case it becomes necessary. The head is then extended. The

next step consists of the removal of the hyoid bone, which gives a much wider space in which to work and facilitates the later repair of the esophagus. The larynx is next skeletonized and the superior cornua of the thyroid cartilages freed. The larynx must be freed down to the esophagus on either side. The trachea is next severed, usually between the first and second rings, as the first ring, if not removed, almost always undergoes necrosis.

Before separating the trachea from the esophagus, the lining membrane of the trachea is injected with novocain to help free it from the esophageal wall. An incision is made through this part of the trachea in the shape of an inverted epiglottis. This tongue of trachea has a twofold function. It acts throughout the rest of the operation as a dam to prevent blood from entering the trachea and it forms a solid wall above the tracheal opening when the wound is closed.

The severed tracheal stump is now sutured to the skin with temporary linen sutures, thus preventing the blood and secretions from seeping into the trachea.

The larynx is next separated from the esophagus from below. A vertical incision is made through the esophagus and the interior is inspected to see if there is any involvement of it by the growth. If so, the next incision, which is made with a curved scissors, should include it with the larynx.

Large Kelly clamps are now placed across the base of the tongue from either side. These pass between the tongue and the epiglottis. The epiglottis is always removed because it is a part of the larynx. All bleeding points that have been clamped are now fulgurated with the diathermy current. Larger vessels are ligated.

The esophagus is next closed from below upward, with interrupted fine catgut sutures, inverting the raw surfaces. Just before the upper part is closed, the feeding tube is inserted through the nose and extended down into the esophagus, 4 inches below the lower end of the esophageal opening. The first row of sutures in the wall of the esophagus is now buried by a second row. When this closure is completed, the temporary sutures holding the tracheal stump are cut, and the tracheal stump is sutured to the skin with 8 or 10 heavy linen sutures.

The wound is packed with 4 strips of iodoform gauze, one strip going upward and outward on the left side, and one upward and outward on the right. The lower drains are inserted downward on either side. These are the four pockets left when the larynx is removed and drains must be kept in them until suppuration ceases. The counter openings on either side of the incision are no longer used, as 2 inches of the wound are left open for drainage.

The tracheotomy tube, which is wrapped with gauze covered with bismuth paste to keep secretions from seeping into the trachea, is now put in place. The tracheotomy tube has an extension of about 2 inches to allow for the dressings over the wound.

POSTOPERATIVE CARE

During the first six hours following operation the patient is given water and orange juice through the feeding tube. Sodium sulfadiazine (15 grains every four hours) is started and continued for a week or ten days. The following day the diet is increased to include orange juice and clear soups. On the second day following the day of operation the patient is put on a high caloric diet consisting of oatmeal, gruel, sugar, milk, cocoa, malted milk, beef juice, eggs, cream and soup.

On the second day following the day of operation, a blood sugar estimation

is obtained. Many patients will show high blood sugar values, which may be owing to interference with the balance of hormonal secretions caused by manipulation and severence of the thyroid isthmus. If the blood sugar is high, the patient is given insulin and in a few days the blood sugar returns to a normal level.

The wound is dressed daily starting on the first day following the day of operation. The four iodoform drains are removed, secretions are aspirated and iodoform gauze plugs are again inserted into the wound. The sutures around the tracheal opening are removed on the fourth or fifth day following the day of operation. We have tried locally in the wound all of the different sulfonamides in an attempt to diminish infection but have returned to the use of iodoform gauze, which seems to be more effective in keeping the wound clean.

The patient is allowed out of bed on the third day.

The condition of the posterior or noncartilaginous part of the trachea is our guide to the time when the feeding tube should be removed and the wound closed. When this area is securely healed the tube may be removed without fear of complications. If there is a fistula in the eosophagus above the trachea, it heals quickly when the tube is removed and the midline incision is pulled tightly together with strips of adhesive. A pressure bandage is put over this area, and the patient is instructed to press over it with his hand when swallowing for the first few days.

SUMMARY

This review of the advance made in the treatment of cancer of the larynx during the past seventy years may be described as a story of battles won but victory still to be achieved. An important battle is now in progress which, with good fortune, will bring us much nearer to the final victory. This battle is our effort to expand and make more effective our plan of education in regard to cancer of the larynx. As physicians we must remember at all times that we are teachers and that the burden of instructing the community in the subject of cancer remains with us. Many agencies are active in this educational work and many more have the facilities for it, and are eager and waiting to be used for this purpose. Service clubs, parent-teacher organizations and study clubs will welcome the news of cancer that can be cured.

Medical students, house officers and internes should be instructed in the use of the laryngeal mirror. Students must be directed to make the mirror examination of the larynx a part of the routine physical examination and last and of greatest importance, the laryngologist is urged to examine the larynx of every patient, keeping in mind that laryngeal cancer in certain locations will not cause pain, will not produce a hoarse voice and wil not elicit a complaint from the patient. By ceaseless reiteration to the public of the significance of the hoarse voice and by constant insistence to physicians of the importance of a mirror examination of the larynx as part of physical examinations, we will soon see effective results from our efforts. This educational plan will do much to aid in bringing to the laryngologist the incipient cases of cancer of the larynx, the patients who can qualify for the operation of laryngofissure which gives more than 80 per cent cures.

DISCUSSION

L. B. BERNHEIMER, M.D., Chicago, Ill.: Dr. Cunning's paper has been so complete that he has left very little for a discusser. However, there are two points on which I believe there can be controversy. One is his statement concerning the local use of the sulfonamides and the other is his statement that roentgen

therapy of laryngeal tumors is still in an experimental stage.

In regard to the local application of sulfonamides, it has been my experience that the local application of sulfathiazole powder, combined with pre-and postoperative oral administration of sulfadiazine has resulted in more rapid primary healing than in those instances where oral administration was relied upon alone. As a matter of fact, my confidence in such combined chemotherapy has become so great that I have closed three laryngectomies without drains and have really found that healing was amazingly rapid, the feeding tube being removed the fifth day following the day of operation; the patient leaving the hospital on the seventh.

I am not foolish enough, however, to recommend closure of a laryngectomy without drainage as a routine procedure. The patients in whom this was done were watched very closely, and I can say that the anxiety that went along with this procedure certainly was not worth the end result.

Concerning irradiation of laryngeal carcinoma, I may say that I have had the good fortune to be associated in a large tumor clinic in which there exists an extremely critical but friendly cooperation between the departments of radiology and surgery. Indications for treatment of lesions by either irradiation or surgery have been evolved by this cooperation and at present our guides are briefly as follows: (1) Intrinsic carcinoma of the larynx with freely movable laryngeal structures are considered non-infiltrating and are usually treated with external irradiation. These lesions are frequently anaplastic, but the clinical aspects of the neoplasm as manifested by motility have proven to be a more important factor in determining selection of a type of therapy than has cytological studies of the growth. The end results of x-ray treatments of such lesions over a period of five years have proven to be equal to those obtained by surgical removal of such growths by laryngofissure and cordectomy.

(2) Intrinsic epidermoid malignancy of the laryngeal structures is considered infiltrating and is treated surgically by laryngectomy. Such infiltrating lesions are frequently adult types, but again motility has proven to be a more satisfactory guide than histo-pathologic pictures.

(3) Primary subglottic lesions are usually treated surgically. The anatomic location of such neoplasms seems to influence unfavorably the cellular response of such tumors to irradiation.

(4) Extrinsic lesions, if too extensive for surgery, are irradiated, but frequently irradiation is combined with such surgical procedures as block neck dissection or even laryngectomy after regression of the primary lesion.

(5) Intercurrent upper respiratory infection during irradiation therapy apparently modifies cellular response unfavorably. In the face of such an episode x-ray therapy may be stopped and surgical intervention instigated.

(6) Failure of response of an irradiated laryngeal neoplasm does not contra-indicate postirradiation surgery. Such dissections are frequently more difficult and healing is often delayed. However, we have performed a number of laryngectomies on patients who have had a full course of x-ray treatment, up to six or seven thousand roentgen units, and with chemotherapy, usually have closures without fistula formation.

I certainly agree with Dr. Cunning's statement that "Credit without limit must go to the laryngologist who achives a cure of a small epithelioma of the vocal cord by irradiation therapy."

LEROY A. SCHALL, M.D., Boston, Mass.: The paper of Dr. Cunning's on the "Advances in the Treatment of Cancer of the Larynx" contains much that meets with our approval and much that is very instructive. No one who deals with cancer can disagree with his summary; that we must conduct an educational program not only to the laity but to the physician, and that it should start with the instruction of the medical student in the proper use of instruments and the realization that an examination of the larynx should be done on every patient.

I envy Dr. Cunning his surgical team in these days of shortage of physicians. It is ideal to have such a team and such a team does cut down the hard work and the time required for total laryngectomy. I agree most heartily with the preoperative preparation of the patient and with his postoperative care. I like sulfonamides locally in the wound after operation. I wish he had said something about the hygiene of the mouth before operation, but I know that he practices hygiene of the mouth on his patients.

We are happy to hear the statement that fixation of the vocal cord means infiltration of the tumor. Too long have I heard that it means infection, and I am happy to hear, too, that the grading of the tumor helps to decide

on treatment. I agree with Dr. Cunning that many factors enter into the decision as to treatment and that each case is a highly individual one. I have never been accused of not cooperating with the radiologist. I know that there are certain tumors that irradiation can cure. The radiologists with whom I am associated have a friendly, cooperative attitude, and we have arrived, through trial and error, at fairly definite conclusions.

Total laryngectomy can be performed perfectly easily under local anesthesia. I happen to prefer a general anesthetic. I do not like tracheotomy. For many years I have used a breathing tube, put in as soon as the patient is relaxed, and that calls for a resident to be with the patient from the time the anesthesia is started until the tube is in.

In our surgical experience, there are certain well-defined surgical principles that we must not forget. When I have my larynx removed for cancer, I wish the surgeon who is doing it would forget all about conservatism. I would like him to lay my neck wide open. I would like him to examine the glands along the carotid sheath and particularly at the bifurcation of the carotid artery. We know from block neck dissections that glands that are not palpable show an incidence of metastasis of about 10 per cent. I cannot get away from Sir St. Clair Thompson's teachings, and I like an incision which permits a wide exposure of the neck. Then, too, I would like the pretracheal muscles to be removed. I do not wish the surgeon to rely on hope or chance that these pretracheal muscles, which are rich lymphatic beds, do not already contain cancerous cells. Then I would like the midline of my neck to be closed tight with drainage away from the midline. I do not want the tracheal orifice to be the cesspool for a sewer draining to it from above.

I recognize, however, that a procedure which gives good results is the procedure of choice, and I have no quarrel with the person who produces results by a procedure that he prefers to mine. It so happens that I prefer a wide exposure.

CHEVALIER L. JACKSON, M.D., Philadelphia, Pa.: I am sorry that it would not be appropriate for me to start out by discussing Dr. Schall's discussion of narrow field laryngectomy. However, I do thank him for it and I hope to discuss his discussion at some future time.

The subject of classification and selection of treatment is very interesting to me, and particularly the pathological classification. Dr. Cunning's series showed an overwhelming predominance of the grade 2 tumors, but, as he pointed out and as Dr. Eggston pointed out, no two pathologists would balance up quite the same in even the same series of cases. In our series also, grade 2 tumors do largely predominate, but only to the extent of 44 per cent as compared with the 35 per cent of the cases which were considered grade 3. Apparently our pathologists are dividing off some of what Dr. Eggston would call grade 2 cases and putting them among the grade 3 cases. I think that this point is of importance, because in our analysis of cases, we find that the five-year cures are much higher in percentage in the grade 2 than in the grade 3 cases according to our own grading. In a series in which the grade 2 and the grade 3 were of about the same number, we had a 73 per cent cure of grade 2 cases treated by laryngectomy and irradiation, whereas in the grade 3 cases we had only a 46 per cent cure rate.

Of course, no one series is to be taken too seriously, but I feel that these figures should go on record, and we are continuing to study our cases with careful attention to the histologic character of the tumor, because, regardless of the importance of motility of the cords and the location and extent of the lesion which, I, like Dr. Cunning, regard as a very important factor, it is inescapable that the study of the tumor under the microscope must be recorded and studied and analyzed. In our series the grade 2 cases showed much better results than those of grade 3, and that held for both those treated by surgery and those treated by x-ray. The ones treated by surgery were a little better than those treated by irradiation, however.

Another point in Dr. Cunning's paper I should like briefly to comment on is the question of indications for laryngofissure and whether or not laryngofissure can be done in cases where the lesion reaches the anterior commissure. In a series of 310 cases treated surgically, between 1930 and 1937, laryngofissure was done in 132 cases and in 178 cases laryngectomy (42 per cent laryngofissure and 57 per cent laryngectomy). Of course, I realize that that relative percentage is to be considered with the full understanding that some of these patients were sent to our clinic because it has been associated particularly with the operation of laryngofissure, so that it does not mean that if Dr. Cunning had seen these patients he would not have done laryngofissures in many more than he has in the series.

On the question of anterior commissure lesions, in 99 patients operated upon between 1930 and 1940, a ten-year period, there were recurrences in 15, or an 85 per cent cure. Of those 15 recurrences, 18 per cent were anterior commissure and 16 per cent were restricted to the middle of the cord.

I do not believe I have time to comment upon treatment by the roentgen ray, because I want to say a few words about the technic of laryngectomy. It is our experience also that fistula is becoming a more and more rare complication of the operation of laryngectomy. This is due partly, we think, to the attention given to preoperative care, as it doubtless is in Dr. Cunning's experience and in the experience of others, and the minimizing of infection by increased attention to hygiene of the mouth. I mentioned in connection with my film on laryngectomy, shown yesterday, that it is routine in our clinic to have an O.K. by the dentist on every patient before he is subjected to laryngectomy. There is no point in rushing the patient to the table and getting on with the operation of laryngectomy while the mouth is dirty, because even a week to ten days can make all the difference in the world in the condition of the mouth, and that time would very rarely make any difference in the prospective cure by the operation.

For anesthesia we, like Dr. Schall, prefer general anesthesia, but we use avertin and intravenous pentothal by preference to other forms of generalized anesthesia.

On the question of drainage, I must say that I am delighted to heard that Dr. Cunning has come to use simple drainage through the midline. His iodoform wicks go up into the four corners of his wound, and I believe that they will take care of all drainage; if, in the occasional case, drainage laterally is necessary, I think it can be done later. Our experience in the series of cases that I referred to yesterday, some 57 patients operated upon in the past two and one-half to three years, has led us to believe that we are fully justified in continuing to drain through the midline. We use a metal or glass sump drain, rather than gauze, and we sometime use two sump drains, turning them in the two lateral directions, and we have had no difficulty in keeping the drainage from flowing into the tracheal stoma.

In the series of cases to which I refer there have been no postoperative mortalities, which does not mean, as I said yesterday, that that sort of success will continue for the next week or the next month; it is just merely a statement of fact, that that consecutive series of cases was without operative mortality.

Finally, I would like to add my emphasis to what Dr. Cunning has said about continuing our fight for education of the laity and the profession. It is true we will make some neurotic patients too cancer conscious. We will have them coming in to let us see their larynges more often than we care to, and we will cause them some sleepless nights, but the great majority of people will not be so affected. They will take sufficient notice of what we say, to go to their physicians often enough to have suggestive symptoms investigated and early diagnoses made. This idea of Dr. Cunning's, that we should insist on the laryngeal mirror examination as part of the routine physical examination, I agree, is an excellent one. I think this teaching should be spread through all the medical schools, and in this way knowledge and skill in the technic of mirror laryngoscopy would inevitably be developed and disseminated.

I should like to say that it is a privilege to discuss Dr. Cunning's paper, as it has been a privilege to see him work in his clinic at the Manhattan Eye, Ear and Throat Hospital.

DANIEL S. CUNNING, M.D., New York, N. Y.: First of all I want to thank these three gentlemen who so nicely discussed this paper.

I do not believe that the type of anesthesia or the narrow or wide field incision makes much difference in the end result. We have been doing what we call the narrow field, the straight incision, for two or three years. I had never heard the expression until yesterday when I saw the movie. I think you can open up the neck just as wide as you wish with the "T" or straight incision.

As to the hygiene of the mouth, of course, we always use that. When I started preparing this paper two or three months ago, I discovered I had 140 or 150 pages and I have been the rest of the summer trying to cut it down to a twenty minute discussion and that is why a lot had to be skipped. The main thing that I am trying to put over in this paper is early diagnosis, and I do not believe we are doing enough to emphasize this point.

ANESTHESIA IN OTOLARYNGOLOGY

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BY INVITATION

STRENUOUS and resolute efforts to improve and extend the scope of surgical therapy until recently have been hampered and delayed by the coat-tail clinging stepchild, anesthesia. Although introduced by a dentist, William Morton, and a physician, Crawford Long, and treated early as a scientific branch of medicine by John Snow of England and James Gwathmey of this country, anesthesia soon fell into the hands of technicians. Progress was limited largely to technical improvements and empirical changes in an effort to keep pace with the rapid advances in surgery. In the past, the surgeon was judged solely by the dispatch with which an operation could be performed. The surgeon has long realized, however, that uniformly good results are dependent, not solely upon the dexterous execution of the technical aspects of surgery, but are dependent in large measure also upon an appreciation of the alterations in the physiology of the patient, occasioned by the pathology present and the surgical manipulations.

Recent advances in anesthesiology indicate an appreciation by anesthesiologists of the need for complete knowledge of the fundamentals of physiology and pharmacology associated with the application of anesthetic drugs. No longer should the anesthetist be judged solely by his ability to keep a patient narcotized. The surgeon may expect the anesthesiologist to fulfill several functions which contribute to the convenience of the surgeon and the comfort and safety of the patient. These functions, as they pertain to otolaryngological surgery, are (1) premedication (2) selection of agent and technic (3) technical application and (4) control of drug depression or reaction.

Premedication is a misnomer. The nonvolatile drugs administered to the patient preoperatively are actually a part of the anesthesia. They possess definite pharmacological actions and must be administered as intelligently as any other anesthetic agent. The success or failure of many infiltration, regional or topical anesthetic procedures is contingent upon proper preanesthetic medication. The safety and stability of general anesthesia is also directly influenced by the premedication. The use of routine combinations and dosages is condemned in favor of individualized selection on the basis of the patient's physical state and the effects desired.

A cooperative and calm individual is more amenable to regional, infiltration and topical anesthesia. The forcible application of local anesthesia on the uncooperative patient frequently results in poor anesthesia, inadequate operating conditions, and may terminate tragically. If these anesthetic technics are to be used, the drugs administered preanesthetically should provide hypnosis, protection against the toxic effects of the anesthetic drug, analgesia, reduction of reflex irritability and amnesia. Such effects can be accomplished by using barbiturates for hypnosis and protection against the toxic effects of cocaine or similar drugs, morphine for the reduction of reflex irritability and analgesia, and scopolamine for amnesia. The an-

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esthesiologist can be of service to the otolaryngologist by assisting him in the selection of appropriate drugs and dosages. He may also accommodate the surgeon by the intravenous complementation of morphine and scopolamine in cases where the dose prescribed was insufficient, or in prolonged operations during which the effect of original premedication has worn off.

Premedicating drugs should also be ordered prior to inhalation or other technics of anesthesia with direct reference to the particular agent to be employed. In the same individual, different drugs and dosages should be employed before cyclopropane, nitrous oxide or pentothal anesthesia. The premedicating drugs have no effect on the surgery only insofar as they aid or hinder the anesthesia. As a consequence, it is the function and duty of the anesthesiologist to prescribe preanesthetic medication.

Now, how does this theory work out in practice to your and the patient's advantage? What difference does it make whether or not a patient gets the right drugs at the right time? It means that a patient coming to the operating room properly prepared for a bronchoscopy will be tranquil, relaxed, cooperative, protected against some of the toxic effects of cocaine, and perhaps blessed with amnesia. It means a satisfactory procedure quickly and deftly completed without constant berating and restraint of the patient. It means that the ordinary middle-aged adult will receive, instead of the usual 1/4 grain of morphine and 1/150 grain of atropine given hypodermically as the patient passes the floor nurse's desk on his way to the operating room, 3 grains of nembutal or seconal 2 hours before operation and 1/4 grain of morphine and 1/100 grain of scopolamine one and a half hours before operation. He will then be dozing when he passes the nurse's desk instead of fidgeting on the cart. The barbiturates are given that long beforehand to permit passage of the capsule through the pylorus before the morphine constricts it. The morphine and scopolamine are given that far ahead because it takes that long for morphine to reach its maximal action when given subcutaneously.

Another example of the value of competent preanesthetic medication is its use in tonsillectomies in children under ether anesthesia. Too many times a 10-year old is brought to the operating room sitting up on the cart, crying, frightened half to death, with distraught parents clinging to his hands and patting his hair. He usually has had 1/12 grain of morphine and 1/300 grain of atropine subcutaneously on the way to the operating room. He is well prepared to give a real tussle during induction, have jerky, uneven respiration throughout anesthesia, and he will retain very unpleasant memories of the whole procedure. He is entitled to 1/6 grain of morphine and 1/150 grain of scopolamine one and a half hours before anesthesia. With this dosage he would ride to the operating room awake but markedly less apprehensive. His induction would be smooth, his anesthesia even, and his memories vague.

In order to select the most suitable agent and technic to be used in each individual case, the anesthesiologist must first learn the requirements of the surgeon. These vary with each surgeon and with each operation. For instance, it is advisable to know the degree of relaxation desired and whether or not the endotherm is to be used. If the surgical manipulations are to be near nerve plexi, he must know the manifestations of those reflexes and whether they should be retained or obtunded. He must know whether or not it is advisable from the surgical standpoint to have the patient regain his protective reflexes promptly. He must know the surgical approach

and the optimum position of the surgical team so that he and his equipment can be removed from that site. In essence, the surgeon need only state the desired requirements and the anesthesiologist is obligated to fulfill them without sacrificing the safety of the patient. Fortunately, it is now possible with the application of present day anesthetic agents and methods to achieve mutually satisfactory working conditions.

In order that I may illustrate this practically, let me presume the case of a healthy 45 year old man on whom a laryngectomy or a laryngeal fissure is to be done. If the patient is adamant and refuses to have the procedure done under local or regional anesthesia, an agent must be used that will provide complete narcosis with safety for the patient and convenience for the surgeon. The surgeon may desire to use the endotherm and he wants to have room to work unhampered by anesthesia equipment and anesthetist. In this event, one could use avertin and nitrous oxide, nitrous oxide and oxygen alone, or pentothal. In any case, the patient will need to be intubated. This can be done by transnasal or transoral endotracheal catheterization which may be supplanted later, as the trachea is severed, by a tube passed into the distal end of the cut trachea. By using a tracheal catheter, the anesthetist can be removed from the field as will be shown later.

If the patient is willing, the whole surgical procedure can be done under cervical nerve block. In this case, it is necessary to complement the block by topical application of cocaine in the trachea and by injection of procaine into the superior laryngeal and glossopharyngeal areas. The cervical block may be complemented after severing of the trachea by pentothal anesthesia. This expedites the surgery by eliminating the time necessary for complemental blocking of nerves not included in the cervical block. Although there are innumerable other examples that could be cited, it is evident from the solution of this problem in anesthesia that there are a number of individual agents and a number of combinations of agents and technics available for a satisfactory anesthesia.

I have mentioned pentothal sodium in discussing agents for laryngectomy. If I may digress a minute, let me say that intravenous barbiturate anesthesia has proved to be a substantial part of the anesthetist's armamentarium. May I emphasize, however, that it can also, when imprudently used, be a distinct hazard in otolaryngological work. At first consideration, otolaryngologists often think that any drug not administered by the inhalation route is particularly suited for their type of surgery. They feel that by using such a drug as pentothal sodium, introduced intravenously, the anesthetist and his gadgets can be removed from the field. Such is not the case if these drugs are to be used safely. In inhalation anesthesia, the equipment is there, not only to administer the agent, but also to maintain the airway. It is as important and perhaps even more important to establish and maintain an airway with barbiturate anesthesia as it is with ether, consequently, as much equipment will be necessary for pentothal anesthesia as for inhalation anesthesia. The barbiturates have the property of enhancing the laryngeal reflexes and any direct or reflex excitation predisposes to laryngospasm, coughing, sneezing, or hiccoughing. It is therefore advisable to use pentothal in otolaryngologcial surgery only when there is to be no contamination of the pharynx with blood, pus, or mucous, unless the patient is intubated. The intubation should be done with the patient awake and the throat and larynx cocainized. Instrumentation of any sort which involves the vocal cords causes frequent prompt and se-

vere laryngospasm, which has, at times, necessitated tracheotomy. I realize that this is restricting the use of a convenient and pleasant drug. I realize, also, that many of you have used it in hundreds of cases with little or no difficulty without respecting these safeguards. I know, however, that unless these safeguards are used, serious trouble will eventually result from the indiscriminate use of pentothal. It may not happen until after use in 3000 cases, which, roughly speaking, is three pentothal anesthesias every day for three years, but one death from anesthesia in 3000 cases is a very high mortality.

The technical administration of anesthesia is a function which reflects the anesthesiologist's knowledge of the fundamental factors affecting that anesthesia. The "giving of an anesthetic" is no longer a matter of routine or rote, but a continuous evaluation of the patient's responses to anesthesia and surgery. This may lead to a change of agent, a change of technic or a change in depth of anesthesia. Because of his close supervision of the patient's physical state, the anesthesiologist is first aware of the need for supportive measures in the event of impending shock and, on occasions, can best determine the patient's ability to withstand an extension of the surgery. He should be prepared to impart the details influencing these decisions. The surgeon is engrossed in the surgical procedure, and his mind should be free to handle his own problems.

In otolaryngological surgery, the principal problem in the technical administration of anesthesia is the establishment and maintenance of a free airway without hampering the operator. No one single factor is more important in good anesthesia than the airway. The lack of an adequate airway is the responsible factor for a large percentage of poor anesthesias and for the vast majority of anesthetic fatalities. It is an interesting paradox that in civil life the patient is given protection by law for the maintenance of his airway, whereas in many hospitals his airway is seriously jeopardized with indifferent protection under the guise of anesthesia. The maintenance of an airway during otolaryngological surgery can, in many cases, be accomplished by simple measures. It is, however, frequently necessary to utilize the endotracheal catheter.

The anesthesiologist and the surgeon must not forget that in many patients being operated upon under infiltration, regional or topical anesthetic technics, attention to the airway is also important. Many of these patients have their noses and throats full of packs, their faces covered with draperies upon which the second assistant leans, and their heads and necks in awkward positions. These patients are not infrequently in dire need of a little police protection of their airways.

Otolaryngologists use cocaine or similar drugs frequently. Reactions to these drugs are not uncommon. A surprising number of otolaryngologists are uninformed or misinformed about the treatment of these reactions. The anesthesiologist can be of service in emergencies of this sort and should be prepared to institute prompt and thorough treatment.

Reactions to cocaine or similar drugs are in 99 per cent of the cases due, *not* to drug idiosyncrasy or sensitiveness, but due to the presence of the drug in the circulating blood in sufficient concentration to depress the vital functions of the brain stem. Many of you have had the experience of having a patient react to cocaine at a second application. I do not believe this can be interpreted as the development of an allergic sensitization from the first application, but rather that a larger amount was absorbed into the blood stream on the second use of the drug. Substitution

of other topical or infiltration agents will not always eliminate the danger of reaction, since in effective percentages and concentrations all of these drugs have the ability to produce reaction. Some surgeons, for instance, have changed to pontocaine following an unfortunate experience with cocaine. It is true that pontocaine is effective in 1 and 2 per cent concentrations, but it is, however, about five times as toxic as cocaine and therefore is comparable to a 5-10 per cent solution of cocaine. Also it lacks the vasoconstricting function of cocaine, and consequently might be absorbed more rapidly and therefore be more prone to produce reactions.

If one remembers that reactions to these drugs are fundamentally due to the amount present in the blood stream at any one time, he can use the drugs rationally with comparative safety, and treat the reactions logically.

Prophylactic treatment of reactions to cocaine or similar drugs includes the use of a barbiturate as premedication. The barbiturate depresses the hypothalamus and helps to minimize convulsant features of drug reactions. Whenever these drugs are applied to areas where absorption is rapid, the concentration must be as low as possible and still effect good ansethesia, and it should be applied slowly.

The reactions to cocaine and drugs of similar action are usually of two types, immediate and delayed.

The immediate type of reaction is caused by the sudden absorption of a large amount of the drug in a short time, such as accidental intravenous injection or the application of a high concentration to a vascular area. It is manifest by prompt and complete circulatory and respiratory collapse. Immediate artificial respiration with oxygen and intravenous use of ephedrine or neosynephrin to elevate the blood pressure may save the lives of a few of these individuals. Intracardiac administration of adrenalin may also be a life saver. Ephedrine or neosynephrin should be used intravenously until the blood pressure is restored to within 10 per cent of normal. Usually 15 mg. of ephedrine or 5 minims of neosynephrin can be given intravenously at ten-minute intervals until results are obtained.

The delayed type of reaction is due to a slower build-up of a toxic level of the drugs in the blood stream of the patient. In this type of reaction the patient becomes stuporous, the pulse becomes slower, the blood pressure falls, and twitchings or convulsions may develop with respiratory failure. Oxygen is again of benefit with artificial respiration, if necessary. Ephedrine or neosynephrin may be used if the blood pressure falls. If convulsions develop, a barbiturate, such as sodium amytal or pentothal sodium, introduced intravenously, will check them. The barbiturate should be given until the convulsion is stopped. The breathing may also stop, but such stoppage can be easily remedied by artificial respiration. The majority of patients developing this latter type of reaction can be saved, provided prompt, complete physiologic and pharmacologic methods are used. All otolaryngologists should have oxygen, with a means for applying it, immediately available together with ephedrine or neosynephrin and an intravenous barbiturate. You will note that I have neglected to mention coramine, metrazol, caffeine, hot coffee enemas, rectal dilatation, etc. I do this deliberately: They have no place in the treatment of reactions to cocaine or similar drugs. When adrenalin is used in combination with procaine, it is important to differentiate between adrenalin reaction and procaine reaction. As opposed to the stuporous patient with a slow pulse and low blood pressure in procaine poisoning, the patient with an overdose of ad-

renalin will be anxious, have palpitation, tachycardia and a high blood pressure.

Another interesting and new example of the cooperation that can be manifest between anesthesiologist and surgeon is the recent introduction of the use of curare for muscular relaxation during endoscopy. Curare has no analgesic or anesthetic properties. It does, however, provide excellent muscular relaxation. I have used curare during inhalation anesthesia for abdominal muscle relaxation with excellent results. Its application during endoscopy came about as a result of a search for a satisfactory answer to the problem of the occasional individual who either could not or would not relax his jaws and neck sufficiently to permit good endoscopic examination. In such individuals it was necessary to provide complete narcosis by one of several methods. This often was not satisfactory for the endoscopist, nor safe for the patient. By using topical anesthesia in combination with curare in a patient well premedicated, the relaxation is excellent, the examination can be completed with ease, and the patient is reasonably comfortable and spared the hazards of general anesthesia for such a procedure. The curare is usually given intravenously and the effect is obtained promptly. Its action wears off in about twenty minutes. Occasional laryngospasm is encountered, but this can be eliminated or minimized by adequate cocainization. Respiratory depression occurs at times, but is usually not sufficient to embarrass the patient. If it is, brief artificial respiration with oxygen soon restores adequate function and the procedure can be completed without haste. Curare is recommended only for the difficult patient, who, fortunately, appears infrequently, as a substitute for general anesthesia. Preliminary observations show that it is satisfactory, safe, more convenient to the surgeon, and pleasant for the patient.

It has been possible in this brief period to give only an inkling of what can be done by the modern anesthesiologist for the otolaryngologist. The discussion should point out that the functions of the anesthesiologist are the functions of a scientist and a physician. As in all branches of medicine, he endeavors to restore the patient to normal. The scientific rather than empirical approach to the problem of anesthesia in otolaryngology is reflected in lower morbidity and mortality and greater convenience to the surgeon. The stepchild, anesthesia, now has grown and awakened to his responsibilities, and can walk forward beside the surgeon in the progress of medicine.

DISCUSSION

Voss Harrell, M.D., Detroit, Michigan: Anesthesia is not the stepchild to the surgeon as quoted but, I believe, a full cooperating partner. We all know that in unskilled hands the grief resulting from faulty anesthesia is eventually heaped upon the shoulders of the surgeon. Improvements both in surgery and also anesthesia go hand in hand and should share alike. To me cooperation with the anesthesia department exists before, during and after surgery; we always attempt to plan together. It is my custom always to ask the anesthetists what preoperative drugs they prefer. I less often inquire in regard to postoperative drugs. In other words to me teamwork is most important.

In reference to our greatest find of recent years, sodium pentothal, our routine has been preoperative medication with morphine sulfate (¼ grain) and atropine sulfate (1/50 grain) in all nose cases. On patients whose tonsils and adenoids have been removed we often use the same medication after the tonsils are out and the patient is well anesthetized so as not to depress respiration. We start using a 5 per cent solution of sodium pentothal and when the patient is anesthetized continue with a 2 per cent solution of sodium pentothal. In our hospital we do not give any of the barbiturates two hours before operation but prefer thirty minutes to one hour as we believe giving the patient it two hours

before operation depresses respiration. In children we often give the barbiturates by rectum. We make pin holes in each end of the capsule and introduce it into the rectum either with or without water or saline as preferred. In our work sodium penthothal has been the ideal supplementary drug, even with local anesthesia, as it is so much easier to administer and it is a better anesthetic for the patient. In all our nose cases we cocainize first and follow with sodium pentothal. The preservation of the airway by the use of the tracheal tube has been stressed although we feel it is the duty of the anesthetist to supervise the airway. We have used sodium pentothal by the technic of Thomas in over 200 cases of mastoiditic and otolaryngologic surgery. These include patients with hypertension, diabetes, cardiac failure and blood dyscrasias. We have found it very effective. This technic is, I believe, second choice in patients subjected to tonsillectomy and adenoidectomy, being usable in a few selected patients.

Although the essayist states that intravenous ephredrin can be used in cocaine poisoning, our experience qualifies us to state that one must be very careful in using it where advanced cardiac lesions are present. I personally use oxygen and the barbiturates in anesthetic emergencies at the same time as delay in the use of one or the other may prove fatal.

In our hospital we know nothing of curare. The statement that it will relax the jaw and neck in endoscopy work interests me very much. I will certainly give it a try. In depression of the respiration we use oxygen and carbon dioxide at once. We also use them in any prolonged anesthesia. We also give the patient metrazol on the table if anesthesia with sodium pentothal is of forty minutes duration or longer. We have had 3 patients in whom a distinct personality change followed anesthesia with sodium pentothal lasting over forty minutes. We felt the personality change was owing to anoxemia of the brain.

Dr. Cullen's presentation is timely and well chosen. However in stating that anesthesia is the stepchild of surgery he is not over the barrel with me as without a harmonious wedlock of the surgeon and the anesthestist our future will be disturbed by many unnecessary difficulties.

T. C. GALLOWAY, M.D., Evanston, Illinois: Advances in anesthesia have always led the way to advances in otolaryngologic surgery. Such a fine exposition as we have heard today indicates that such leadership is continuing. On each of three occasions I have listened to Dr. Cullen with increasing interest and profit.

It is an ideal greatly to be desired to have available for all patients, private and institutional, such special training as he has indicated. It has been a great comfort for us at Cook County Hospital to have such service from Dr. Osborne Brown.

Unfortunately it is not possible at present, especially under war stress, to provide even all teaching hospitals with such trained men. The otolaryngologist, if he knows his own limitations and is duly conscious of the dangers, may be able to carry on some of the special technics with nurse anesthesists. This will be so because the laryngologist is trained to expose the larynx and maintain the airway particularly in endotracheal and intravenous anesthesia. If he undertakes this, however, he cannot delegate his responsibility but must add it to that of operating, which is not entirely desirable, and he must appreciate the skill and alertness necessary to get such results as Dr. Cullen has shown.

Dr. Cullen makes these procedures sound almost too easy, and they are undoubtedly easy for him. But I know of two instances, in a relatively small series, of death from intravenous anesthesia with evipal. One was given too casually; the other to a carcinomatous patient with an impaired airway. It is to be hoped that after hearing Dr. Cullen no one will take the procedure too lightly.

Premedication especially with the larger doses Dr. Cullen outlined, has proven increasingly satisfactory. One point to be emphasized is that tactful handling of the patient, and freedom from distraction, movement and noise while the patient is waiting are important in getting the proper effect. In one hospital where these are lacking, I have found the patients more tense and resistive than they would be with no premedication.

Some of us hold the teaching of Chevalier Jackson, that the cough reflex is the guardian of the airway, in such high esteem that we hesitate to give such large doses of sedatives before operations on the throat and larynx, especially when they are to be followed by ether. I am not yet ready to agree to a dose of 1/6 of a grain of morphine and 1/150 of a grain of scopolamine in a 10 year old patient who is to have his tonsils out under general anesthesia and perhaps have his tracheobronchial tree flooded with blood, secretion and crypt debris. I could be convinced as to the safety of the procedure, however, by statistics

on a considerable series of patients so treated which showed that they were free from postoperative atelectasis, lung abscess and bronchiolectasis. Atropine alone with gentle handling and gas induction of anesthesia has, in my experience, been an eminently satisfactory preanesthetic drug.

The warning about the dangers of the substitutes for cocaine is still almost as timely as it was twenty years ago, when I reviewed the literature on this subject for the committee on local anesthesia. It is to be borne in mind that the ratio of toxicity to anesthetic effect of many of the substitutes for cocaine, if they are absorbed into the blood stream, is about the same as for cocaine itself.

STUART C. CULLEN, M.D., Iowa City, Iowa: In answer to Dr. Galloway who still is a little skeptical about our use of 1/6 of a grain of morphine and 1/150 of a grain of scopolamine in 10-year olds, I will say that our series of tonsillectomies and adenoidectomies at Iowa has been free from any postanesthetic pulmonary complications. Because we have not been troubled with postanesthetic respiratory complications in tonsillectomies, we feel justified in affording the 10 year old patients the mental rest obtained from the relatively larger doses of morphine and scopolamine.

T. C. Galloway, M.D., Evanston, Illinois: Do you use carbon dioxide for resuscitation?

Stuart C. Cullen, M.D., Iowa City, Iowa: We do not; we use only 100 per cent oxygen.

THE ROLE OF SARCOID AND OF BRUCELLOSIS IN UVEITIS

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IN A PREVIOUS report on the etiology of uveitis[11] we stated that the importance of sarcoid and brucellosis as etiological factors could not then be properly evaluated. The significance of sarcoid and brucellosis as causes for uveitis was not appreciated until late in this previous study, and search for evidences of these diseases had not been routinely made. The previous study included cases of uveitis admitted to the Wilmer Institute up to July 1, 1939. A survey of the cases of uveitis admitted from July 1, 1939 to June 30, 1943, revealed that during this period there were exactly 200 cases completely studied with the idea of establishing the etiology of the uveal disease. Included in the general study were searches for any evidences of sarcoid and brucellosis. It is the purpose of this paper to present the etiological study of these 200 cases and to discuss the general problems of sarcoid and brucellosis as related to uveitis.

The routine diagnostic procedures in these patients were as follows: 1. Complete eye examination. 2. Medical history and general physical examination. 3. Routine laboratory examinations of urine, haemocytology and blood chemistry. 4. Serologic tests for syphilis. 5. Quantitative intracutaneous tests for tuberculin hypersensitivity. 6. Roentgenological examination of the chest. 7. Routine search for foci of infection in the nose, throat, accessory nasal sinuses, teeth and genito-urinary tracts. 8. Gonococcus complement fixation tests. 9. Agglutination tests for brucellosis, and in some patients complement fixation tests and estimation of the opsonocytophagic index. 10. In cases of suspected sarcoid, biopsy of a lymph gland or skin nodule, estimation of the serum albumin and globulin, and in many cases x-rays of the hands and feet.

There were only 27 case histories in the entire 200 which did not contain this minimum data. These were cases of proven syphilis, sarcoid, herpes simplex, or gonococcal arthritis. In all of these the diagnosis was so clear from the examinations made that irrelevant examinations were omitted. In addition to the above minimum diagnostic procedures, special bacteriological examinations, sensitivity studies, and spinal fluid examinations were carried out when indicated. Frei tests for lymphogranuloma venereum were made in a number of patients, but in no instance did such tests appear of diagnostic value. Protection tests for toxoplasma were not done until 1943.

In this study the criteria for an absolute or presumptive diagnosis of tuberculosis, syphilis, a focus of infection, gonorrhea, and miscellaneous diseases as the etiological factors are exactly those outlined and described in our previous paper. The criterion for the diagnosis of sarcoid as the etiological factor is the histological demonstration of sarcoid lesions in an excised gland or skin nodule. There are no "presumptive" diagnoses of sarcoid The criteria for

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the diagnosis of Brucella infection as the etiological factor are the elimination of other obvious causes for the uveal disease and the finding of serologic or sensitivity evidence of a Brucella infection. It thus follows that all the diagnoses of brucellosis as the etiological factor are in a sense presumptive.

One-third of these 200 patients were private and two-thirds were public ward patients. No strictly out-patients are included in this series. With only an occasional exception, all of the patients were examined and studied under the direction of one of the authors.

The results of this etiological study in these 200 patients are shown in Table I.

As in the former study, the cases reported here are divided into two groups: Group I consists of 102 cases in which the study was so conclusive that an absolute diagnosis appeared justified. Group II consists of 98 cases in which the accumulated evidence was not conclusive. In 76 of these evidence was sufficient to allow a presumptive diagnosis, but in 22 cases the study was either totally unproductive or so inconclusive that no deduction could be drawn as to the cause of the uveitis. These 22 cases are listed as "undetermined." For purposes of comparison the table giving the results reported in our first paper is again reproduced here.

An analysis of Table I and a comparison with the former results shown in Table II yield some interesting observations. The figures for tuberculosis in Table I are 41.0 per cent and in Table II, 49.7 per cent. In our first series (Table II) all sarcoid cases except three were undoubtedly included under tuberculosis. Adding the sarcoid cases to the tuberculosis cases, the incidence of the

TABLE I

Etiology of uveitis, either definite or presumptive, in present series of 200 patients.

	GROUP 1: 102 Patients with Definite Evidence of Etiology		GROUP 2: 98 Patients with Presumptive Evidence of Etiology		TOTAL 200 Patients	
Etiologic Factor	No. of Patients	Percentage *	No. of Patients	Percentage *	No. of Patients	Percentage
Tuberculosis	39	19.5	43	21.5	82	41.0
Syphilis	24	12.0	4	2.0	28	14.0
Sarcoid	15	7.5	0	0.0	15	7.5
Brucellosis	0	0.0	15	7.5	15	7.5
Foci of infection	4	2.0	8	4.0	12	6.0
Rheumatoid arthritis	5	2.5	0	0.0	5	2.5
Gonorrhea	7	3.5	6	3.0	13	6.5
Miscellaneous	8	4.0	0	0.0	8	4.0
Undetermined**	0	0.0	22	11.0	22	11.0
	102	51.0	98	49.0	200	100.0

*Percentages are given in relation to the total 200 cases on which this report is based.

**The 22 cases of undetermined etiology consist of 9 cases of granulomatous uveitis and 13 cases of non-granulomatous uveitis.

combined groups would be 48.5 per cent in the present study, as compared to 50.2 per cent in our former study. The incidence of syphilis in this study, 14 per cent, is slightly higher than in the first study, but this increase is accounted for by the admittance to the wards of a few known syphilitic patients for hyperpyrexia therapy. The figures for gonorrhea as an etiological agent in the two series are statistically comparable — 6.5 per cent and 4.6 per cent. Rheumatoid arthritis is listed in this series as a separate etiological factor, rather than included under "non-granulomatous systemic infection." This is done to make especial note of what appears to us a definite clinical entity, a recurrent serous iritis associated with rheumatoid arthritis and usually a specific hypersensitivity to certain streptococcus antigens.

One glaring difference in the figures now reported in Table I as compared to those previously reported in Table II lies in the number of cases attributed to foci of infection. In this series a total of only 6 per cent of the total cases are attributed to focal infection. Even were the cases attributed to brucellosis grouped under "foci of infection," the total would be only 13.5 per cent. This compares with a total of 26.1 per cent reported in our first series. This difference illustrates clearly our growing disbelief in the importance of focal infection as a frequent cause of uveal disease, and the unwillingness of our consultants to lay stress on trivial and minor foci of infection which appear clinically unrelated to the ocular condition. Consequently, cases of undetermined etiology are much higher in this series.

TABLE II.

Etiology of uveitis, either definite or presumptive, in previously reported series of 562 patients.

	GROUP 1: 244 Patients with Definite Evidence of Etiologic Factor		GROUP 2: 318 Patients with Presumptive Evidence of Etiologic Factor		TOTAL 562 Patients	
Etiologic Factor	No. of Patients	Percentage	No. of Patients	Percentage	No. of Patients	Percentage
Tuberculosis	132	23.5	147	26.1	279	49.7
Syphilis	45	8.0	14	2.5	59	10.5
Sarcoid	3	0.5	0	0.0	3	0.5
Brucellosis	1	0.2	1	0.2	2	0.4
Foci of infection	31	5.5	116	20.6	147	26.1
Gonorrhea	10	1.8	16	2.8	26	4.6
Non-granulomatous systemic disease	14	2.5	19	3.4	33	5.9
Metabolic disease	0	0.0	3	0.5	3	0.5
Miscellaneous	8	1.4	2	0.4	10	1.8
Total	244	43.4	318	56.5	562	100.0

COMMENT

SARCOID: This was the term first used in 1899 by the Norwegian dermatologist, Boeck,[3] who reported upon the skin manifestations of the disease. The changes he described were later shown to be similar to those described in 1889 by Besnier[2] under the name of "lupus pernio." Boeck later changed his first ideas on the relation of these skin changes to sarcoma and termed the condition "benign miliary lupoid." In 1914 the modern concept of this disease was propounded by Schaumann,[24] who recognized the disease to be a chronic granulomatous disorder of uncertain course and spotty distribution, relatively benign, capable of affecting any organ in the body but with considerable predilection for the reticulo-endothelial system. He termed the disease "benign lymphogranulomatosis." The disease is variously known under these and other appelations and is associated with the names of these three authors. The most popular name is the misnomer applied by Boeck — "sarcoid." Within the last few years this disease has received widespread attention and a number of excellent reviews have appeared, notable among which are the reports of Scott[26] (1938), Harrell[13] (1940), and Longcope[17] (1941).

In general sarcoid is a relatively benign chronic disease, occasionally punctuated with acute exacerbations. The constitutional symptoms are slight, and while fever may be present in the early stages of some cases, more usually it is completely absent. The clinical symptoms produced by sarcoid are usually the result of local mechanical obstruction with consequent irritation and interference with function rather than the result of a general toxemia or systemic reaction. A peculiar laboratory finding reported as occurring in about 90 per cent of sarcoid patients is a disturbance in the albumin-globulin ratio in the blood serum due to an elevation of the serum globulin. There is no leucocytosis but sometimes a moderate eosinophilia. The sedimentation rate is usually increased, as is the blood calcium content. Sarcoid patients usually, but not always, show a remarkable anergy to tuberculin, often failing to show any reaction to such large doses as 1 or even 10 milligrams of O.T. injected intracutaneously.

The characteristic pathological lesions of sarcoid are tubercle-like masses of epithelioid cells, usually without any surrounding zone of inflammatory cells or at best a scanty peripheral sprinkling of lymphocytes (fig. 1). There are rarely any evidences of caseation or necrosis. These tubercles usually, but not always, contain giant cells. In 20 per cent of the cases peculiar, hematoxylin staining,

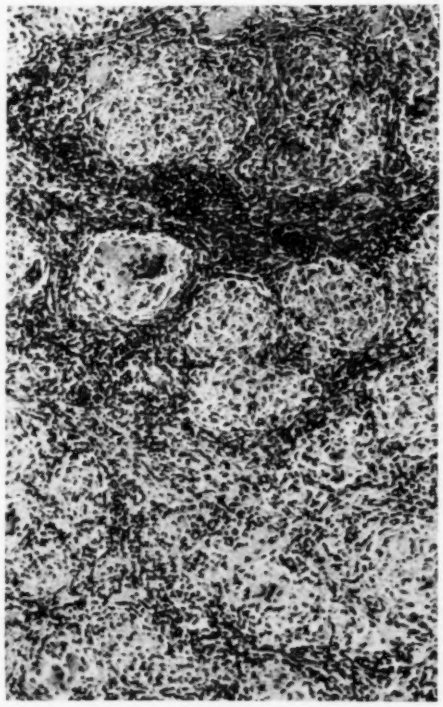

FIG. 1 — Microphotograph of a lymph node from a patient with sarcoid.

usually doubly refractive inclusions known as "Schaumann bodies" are found in the giant cells. If Schaumann bodies are not present it is impossible to differentiate sarcoid lesions histologically from the "hard" tubercles of tuberculosis. In sarcoid, these individual clusters of cells remain small in size but usually become very numerous, forming macroscopic nodules with a proliferative tendency. They may remain unchanged for months or years, usually healing with scanty fibrosis and hyalinization.

While the nodules of sarcoid occur diffusely throughout the body they have a marked predilection for the reticuloendothelial system. A generalized or spotty glandular enlargement is frequent, and the lymph glands practically always show the characteristic microscopic lesion. Roentgenologically visible lesions of the lungs are present in the majority of cases, principally in the form of diffuse infiltration more marked at the hilus and lower portion of the lungs. Nodules of the skin of various sizes and consistency occur in about one-half the cases. The liver and spleen are likewise involved in about 50 per cent of patients with this disease. Less frequent but quite characteristic manifestations of the disease are the rarefaction of the phalanges of the hands and feet, peculiar punched-out areas visible on x-ray examination. Nerve palsies, especially of the facial nerve, occur in the uveo-parotid type of the disease. Lesions of other structures, the heart, muscles, or meninges, are not often clinically evident but are frequently found on microscopic examination. It should be emphasized that while the lesions may be widespread, the disease is a spotty one and the individual patient usually shows only a few of the various clinical manifestations of the disease.

The cause of sarcoid is unknown. The elevation of the serum globulin, the antibody-containing protein of the blood serum, suggests that it may be the result of an actual infection, but no infectious agent, either virus or bacterial, has ever been isolated in the disease. On account of the similarity of the pathological lesions to hard tubercles and the fact that about 10 per cent of sarcoid patients later develop frank tuberculosis, the relationship of sarcoid to tuberculosis has repeatedly been suggested, including the possibility sarcoid might be due to an attenuated human strain or some similar mycobacterium such as the avian strain. However, the characteristic caseation and necrosis of tuberculosis are rarely seen in sarcoid lesions, all transmission and inoculation experiments have been fruitless, and anergy rather than hypersensitivity to tuberculin and various strains of mycobacteria[4] is the general rule. The fact that some sarcoid patients later develop tuberculosis is due probably to the widespread involvement of the lungs by sarcoid and consequent impairment of function and of resistance to tuberculosis.

The presumptive diagnosis of sarcoid depends upon the demonstration of the clinical manifestations of the disease, the skin changes, pulmonary infiltration, rarefactions of the bones of the hands and feet, elevation of the serum globulin, and the frequent anergy to tuberculin. Williams and Nickerson[29] demonstrated that biopsied sarcoid material injected beneath the skin of some sarcoid patients produces typical sarcoid nodules. Later investigators demonstrated that any inert material, killed staphylococci, pigment granules, etc., would produce this same effect. This reaction is of some diagnostic value in Negro patients. The absolute and final diagnosis of sarcoid must depend on the demonstration of characteristic sarcoid lesions in biopsied material, usually a lymph gland.

The first report of ocular involvement in sarcoid was by Schumacher[25] (1909), who observed iritis in a recognized case of sarcoid. Within the next decade a sprinkling of such cases was reported. Ophthalmological interest in sarcoid was stimulated by the almost simultaneous reports of Bruins Slot[6] in 1936, of Longcope[18] in 1937, and of Pautrier[22] in 1938, all of whom identified sarcoid with "uveoparotid fever," a syndrome first described by Heerfordt[14] in 1909. Prior to Slot's paper, in 1927 Hamburger and Schaffer[12] had expressed the view that uveoparotid fever and certain cases of Mikulicz's syndrome associated with uveitis were the same fundamental condition. Since the publication of Bruins Slot's, Longcope's and Pautrier's papers it is generally recognized these are both manifestations of sarcoid.

While sarcoid may affect the ocular adnexa as small nodules in the conjunctiva, lids, episcleral tissues and orbit, such lesions are of little importance other than the diagnostic problem they present. The important ocular lesions are those affecting the uveal tract. Notable among the many reports on the ocular complications of sarcoid are those of Pautrier[22] (1938), Walsh[28] (1939), and Levitt[16] (1941). The most characteristic clinical picture is a nodular iritis, quite similar to the nodular iritis of tuberculosis, although the nodules have a tendency to be larger, slightly pinker and to be more vascularized. The iritis is characteristically quite painless. In other cases there may be no macroscopically visible nodules, but the inflammatory reaction is marked. In either type posterior synechia may form and the picture may develop into a generalized uveitis with blurring of the media, secondary clouding of the lens, keratitic deposits, and changes in the posterior uvea. Some cases may progress to secondary glaucoma and finally to phthisis bulbi, but the general tendency is more favorable. The nodules tend to become hyalinized, the inflammatory symptoms disappear, and the entire process may subside without residua. Involvement of the posterior uvea without anterior uveal change is comparatively rare, but several cases have been reported, the ophthalmoscopic picture being small spots or larger nodular exudates in the choroid with slight surrounding inflammatory reaction. Primary infiltration of the cornea does not occur, any corneal clouding being secondary to the anterior uveal disease.

In almost 500 cases of sarcoid reviewed by Osterberg[21] there were only 27 cases of uveitis mentioned specifically, but apparently the eyes were not regularly examined in all cases, most of these cases being collected from the dermatological literature. In contrast, Levitt[16] found that in 100 cases of sarcoid reported since 1936 there were 28 patients with uveitis and 10 patients with phthisis bulbi. In 29 proven cases of sarcoid studied in the Johns Hopkins Hospital within the past four years, 15 or 52 per cent had an active uveitis. This evidence indicates that ocular involvement is one of the most frequent manifestations of sarcoid.

Previous reports give no indication of the statistical importance of sarcoid in patients with uveitis. The incidence of proven sarcoid in this series (7.5 per cent) is undoubtedly too high for all uveitis. Only about one-third of the general run of patients with uveitis seen in the Wilmer Institute are admitted to the hospital for study or treatment. However, during the period covered by this report practically all uveitis patients in whom there was any suspicion of sarcoid were admitted for study and biopsy. Therefore, the incidence of sarcoid in endogenous uveitis as a whole should be about 2 or 3 per cent. The percentage of sarcoid uveitis is probably

TABLE III
Summary of 15 patients with uveitis and histologically proven sarcoid.

Age	Race	Sex	Ocular Involvement	Keratic Precipitates	Iris Nodules	Enlargement of Lymph Nodes	Serum Albumin (%)	Serum Globulin (%)	X-ray Chest	Intracutaneous Tuberculin Reaction (mgms. OT)	X-ray of Hands and Feet	Fever	Skin Lesions	Peripheral Neuritis	Remarks
13	W	M	Bilat. generalized	large	—	generalized	3.3	2.7	+	—10	—	+	+	—	
15	C	F	Bilat. generalized	large	large	none	3.2	5.1	—	+0.01	—	—	—	+ (N VII)	
18	C	M	Unilat. generalized	large	large	few	4.0	3.3	+	+0.01	—	+	—	—	
19	W	M	Bilat. generalized	large	—	few	5.3	2.3	+	—0.001	ND	—	—	—	
21	C	M	Unilat. anterior	small	—	few	4.4	3.3	+	—0.1	ND	—	—	—	
25	C	F	Bilat. generalized	large	—	few	3.6	4.8	+	—3	—	—	—	—	Keratitis also
26	C	M	Bilat. generalized	large	large	few	4.6	3.6	+	+1	ND	—	+	—	
29	W	F	Unilat. generalized	large	large	few	5.0	2.0	+	—10	—	—	—	—	
30	C	M	Bilat. posterior	—	—	generalized	3.9	4.4	—	+0.01	—	+	—	—	Retinal periphlebitis also
30	C	M	Bilat. anterior	small	—	generalized	ND	ND	ND	ND	ND	—	—	—	Enlarged lacrimal glands
31	W	F	Bilat. generalized	large	large	few	ND	ND	—	—1	—	—	—	—	
33	C	F	Bilat. anterior	large	—	none	3.0	4.6	+	+1	ND	+	+	+ (N X & XII)	
47	W	M	Bilat. generalized	large	large	few	ND	ND	+	+10	—	—	—	—	
53	W	F	Bilat. anterior	large	large	none	4.6	2.3	+	—0.1	—	—	—	—	
67	W	M	Bilat. generalized	large	—	few	4.4	2.8	+	+0.1	—	—	—	—	Keratitis also

ND—not done.
Tuberculin reactions if positive (+) listed as smallest dose to which patient was sensitive, or if negative (—) as largest dose to which patient was tested.

much lower in some sections of this country, because Negroes (common in Baltimore!) are much more prone to have sarcoid than are whites.

In the 15 sarcoid cases reported in this paper there was no significant sex distribution, 9 being male and 6, female. There appears, however, to be some predilection of the disease for Negroes, 8 of the patients being colored, a ratio out of all proportion to the general race distribution. This is in accord with other reports. The ages of our patients ranged from 13 to 67 years. The significant data on the 15 patients is shown in Table III.

somewhat more vascularized (fig. 2). They sometimes disappeared without a trace, in contrast to tuberculous iris nodules, which always leave small scars of atrophy where they heal. The 7 cases of iritis without nodules were clinically indistinguishable from the usual picture of chronic granulomatous uveitis. Only 2 of the patients in this series showed any deep keratitis and corneal vascularization. Keratitic deposits were a constant finding in all 14 of the cases with anterior involvement. The deposits were usually large, of the mutton-fat, epithelioid cell type. In only 2 cases were

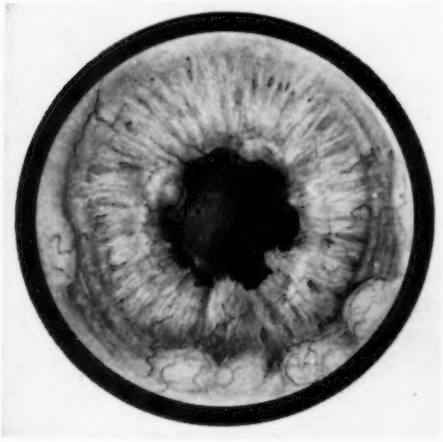

FIG. 2 — Iris nodules in sarcoid uveitis: (a) Active stage

FIG. 2 — Iris nodules in sarcoid uveitis: (b) Subsiding stage in a different patient.

The uveitis in the 15 patients with sarcoid reported in this series was bilateral in 12 patients and unilateral in 3. It was always chronic and often severe. The general tendency was for the entire uveal tract to be involved, although in 3 patients the disease was limited to the iris, and in 1 patient to the choroid. While the most characteristic symptom of ocular sarcoid is nodules in the iris, these were present in only 7 of our cases. The nodules were similar to those described by other authors, slightly larger than those found in tuberculous iritis, more irregular, and small lymphocytic deposits found. In the 1 patient with choroiditis, the lesions were small, poorly outlined, and multiple; and there was some questionable associated periphlebitis.

The general course of the ocular disease was usually less severe than that of tuberculous uveitis, and several of the eyes healed without any residual damage. However, in several cases involvement of the uveal tract was extreme and in 1 patient the eye progressed to phthisis bulbi and was enucleated. Histologically, this eye contained a number of moderately large nodules consisting

FIG. 3 — Microphotograph of sarcoid uveitis:
(a) Low power

FIG. 3 — Microphotograph of sarcoid uveitis:
(b) Medium power.

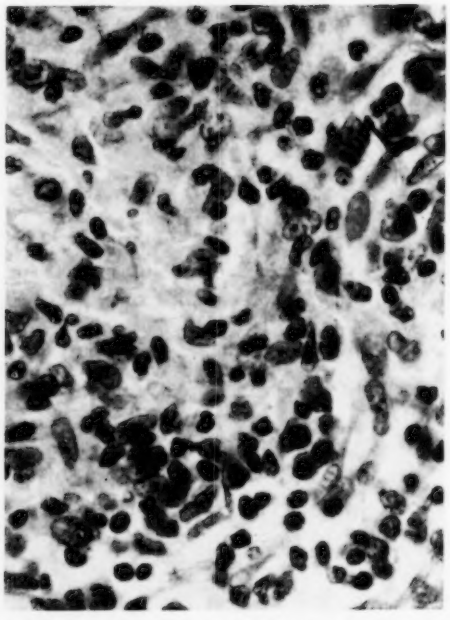

FIG. 3 — Microphotograph of sarcoid uveitis:
(c) High power.

of epithelioid cells and giant cells (fig. 3). No Schaumann bodies were visible. These nodules were most prominent in the peripheral portions of the iris and in the ciliary body. There was also some diffuse infiltration of lymphocytes in the iris and ciliary body, and a very few polymorphonuclear leucocytes. The choroid was normal except for some small accumulations of lymphocytes. The anterior chamber contained considerable blood, and there were extensive anterior and posterior synechiae. The lens was surrounded by a thin cyclitic membrane.

All 15 of these patients showed some systemic manifestations of sarcoidosis. The most frequent lesion was diffuse infiltration of the lungs, this being present in 11 out of 14 patients. Glandular enlargement was present in 12 of the patients but only in 3 was this generalized, in the remaining instances being limited to a few areas, the sub-mental, cervical, inguinal, or axillary glands. It is notable that in the 3 patients with no clinical glandular enlargement, biopsy of an apparently normal gland revealed typical lesions of sarcoid. Three patients only showed skin lesions, and 2 had a peripheral neuritis. Only 4 patients had any fever during the period of observation. In none of the 10 patients who had roentgenograms of the hands and feet were any bone lesions found. While it is generally recognized that the distribution of lesions in sarcoid is spotty, it is nevertheless interesting that in this group of patients the ocular lesions occurred predominantly in association with pulmonary lesions and glandular enlargement, skin and bone lesions being notably few.

Estimation of the serum albumin and globulin were made in 13 of the 15 patients and in only 7 instances was the globulin definitely elevated, over 3.0 per cent.

Estimations of the tuberculin sensitivity were made in 14 of these patients. Only 3 patients reacted to 0.01 mgm. of tuberculin, while 5 were completely insensitive to 1.0 mgm. In several of these patients greater amounts of tuberculin were given. Two patients were completely insensitive to the enormous dose of 10 mgm. and 1 patient showed only a slight reaction to this dose.

BRUCELLOSIS: Brucellosis is also known as undulant fever, melitensis, Malta fever, and Mediterranean fever. The term "brucellosis" is derived from the name of David Bruce[5] who in 1887 isolated the specific organism (Brucella melitensis) from patients with so-called Malta fever. A similar organism (B. abortus) was isolated by Bang[1] in 1897 from cows with infectious abortion. In 1913 a third strain (B. suis) was isolated from swine.[27] The essential identity of all these organisms was shown by Alice Evans[8] in 1918, and subsequently the strains of organisms found in animals were shown to be pathogenic also for man.

In man, the infected individual usually goes through an acute phase of the disease, characterized by fever, sweats, malaise, diffuse muscular pains and aches, loss of weight, and various more or less vague symptoms affecting any portion of the body. This acute attack may subside without sequelae, but usually the patient passes into a chronic phase which may last for years. This is characterized by an intermittent low fever, vague pains affecting almost any part of the body, and lassitude.

During the acute phase of the disease the specific organisms may be isolated from the blood stream, and a high agglutination titer (up to 1:10,000) is shown by the blood serum. As the patient passes into the chronic stage the organisms can no longer be cultivated and the agglutination titer drops, sometimes even falling to zero. A positive complement fixation reaction against the

bacterial antigens of B. melitensis, abortus, or suis usually develops. The diagnostic validity of this reaction is, however, somewhat vitiated by the fact Brucella antigens tend to give false positives or cross reactions with anti-gonococcus serums. As the patient becomes resistant to the disease a high opsonocytophagic index may develop. Most irregular of all is a cutaneous sensitivity to Brucella organisms, which may develop in chronic cases. There is no leucocytosis, but often a lymphocytosis.

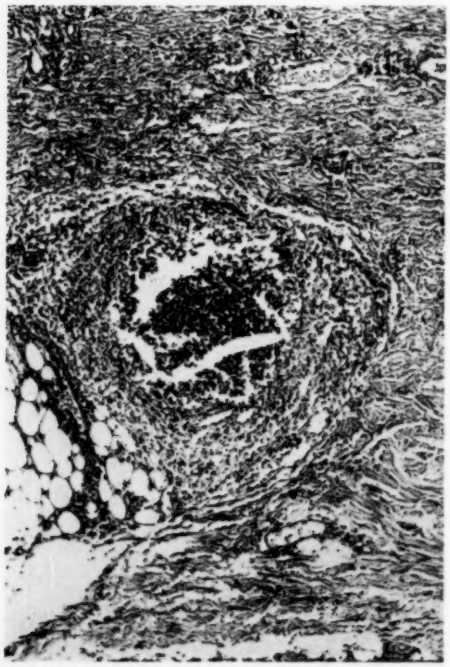

FIG. 4 — Microphotograph of a lymph node from a patient with brucellosis.

The general pathological changes in acute brucellosis are scanty and are probably limited to chronic passive congestion. In chronic Brucella infections there is a proliferation of cells of the reticulo-endothelial system, especially the spleen and lymph nodes, in the form of small nodules (fig. 4). These tiny nodules often resemble tubercles, with epithelioid cells and sometimes giant cells. At times the nodules have necrotic centers with polymorphonuclear cells in and around the necrotic zone. Such central necrosis is not regularly found but when present aids in differentiating the lesion from tuberculosis. The heart occasionally shows a vegetative endocarditis. Nodular reactions in the meninges are common, and occasionally degenerative changes are seen in nerve cells

The diagnosis of brucellosis presents considerable difficulty. In the acute stage it is suggested by the clinical picture and by a history of exposure or the ingestion of raw milk or raw milk products. In the chronic stage the diagnosis is suggested by an unexplained intermittent fever, vague general symptoms with pain in the muscles and joints, lassitude, and by a history of a former more acute phase and of the ingestion of raw milk or its products. The only laboratory tests of any value are the serologic and sensitivity reactions. Since in the chronic stage the agglutination titer is usually low and may even be absent, considerable significance should be attached to weak positive reactions. By using living organisms in the antigen, weak agglutination reactions may be detected which might be missed with a less sensitive antigen. Positive complement fixation tests must be controlled by similar tests against gonococcus antigens. A low opsonocytophagic index has no diagnostic significance and a high index means only the patient has some resistance against the organisms. All these tests, together with demonstrable cutaneous sensitivity against Brucella organisms, only indicate the patient has had the disease. They do not indicate with certainty that the disease is still active, since positive tests may be the residua of an acute infection from which recovery has been made.

The evidence that systemic Brucella infection can cause endogenous ocular

disease is based on experimental and clinical observations. In 1912 Fabyan[9] reported 3 cases of deep keratitis in infected guinea pigs, lymphocytic infiltration and tubercles in the cornea being found histologically. Organisms were recovered from these eyes. In 1928 Orloff,[20] in a comprehensive study of the experimental ocular lesions in guinea pigs, reported that endogenous ocular disease occurred in about 10 per cent of inoculated animals. The affected eyes showed pericorneal congestion, multiple round corneal infiltrates, either central or peripheral, and a definite iritis with exudates over the pupillary space. Histologically, these eyes showed diffuse and focal infiltration of the iris and ciliary body with lymphocytes and epithelioid cells, and only very minor changes in the choroid. Direct inoculation of organisms in the eyes of rabbits (Burky,[7] 1939) produced a generalized kerato-uveitis almost indistinguishable clinically from severe tuberculous lesions. Interest in ocular brucellosis was stimulated by the presentation of bacteriologic and serologic evidence[7] that most horses with periodic ophthalmia (recurrent endogenous uveitis) are infected with Brucella.

The first clinical observation of ocular brucellosis was reported in 1924 by Lemaire,[15] who observed a case of bilateral optic neuritis in a patient with acute brucellosis in whom the organisms were isolated from the spinal fluid. Thereafter, a sprinkling of cases of various types was reported. In 1939 McGinty and Gambrill[19] listed iritis as a recognized complication of brucellosis. In the same year the reported cases were collected by Green,[10] who added 4 cases of his own. In Green's series were 7 cases of non-recurrent uveitis in patients with acute brucellosis. The relative infrequency of uveitis in acute human brucellosis is indicated by this small number found out of hundreds of reported cases of proven brucellosis. Also, Rutherford[23] examined 63 patients with brucellosis ophthalmoscopically and found changes in the discs in 3, but uveitis in none.

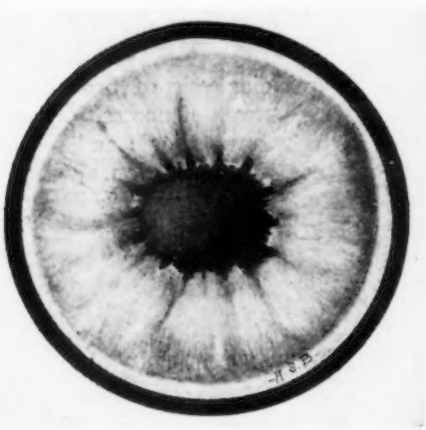

FIG. 5 — Iritis in brucellosis.

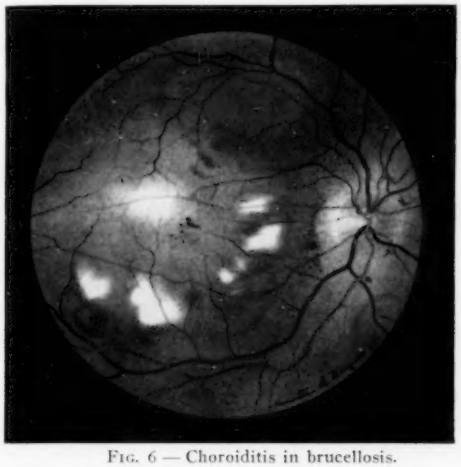

FIG. 6 — Choroiditis in brucellosis.

The ocular picture in the 15 patients in this series in whom the uveal inflammation was attributed to Brucella infection was in no way characteristic or memorable. In 7 of the patients the picture was that of a simple recurrent iritis. In 1 patient there were definite nodules in the iris, in 1 patient ques-

tionable deep nodules (fig. 5) and in a third patient Koeppe nodules were present. In 3 patients the changes were limited to the choroid, the usual picture being one or more elevated, moderately circumscribed exudates with little surrounding reaction or generalized subretinal edema (fig. 6). In 1 patient there was an associated papilledema. In 5 patients the picture was that of a generalized uveitis, in one instance progressing to phthisis bulbi. In 12 patients the uveitis was bilateral, and in 3 cases unilateral. In 11 of the 15 cases the uveitis was recurrent. In 8 patients the uveitis appeared to be of the granulomatous type, with thickening of the iris, posterior synechiae, and heavy epithelioid cell deposits on Descemet's membrane. In 7 patients the uveal inflammation was of the non-granulomatous type without nodular changes in the iris, and with only small lymphocytic keratitic deposits.

In 2 patients there were associated corneal changes. In 1 this was limited to a little peripheral vascularization while in the second the picture was that of a nummular keratitis. In addition to this last case, we have seen 1 other typical nummular keratitis in a patient with chronic brucellosis. The case was not associated with a uveitis and is not included in the series. These 2 cases, however, bring up the question if there might be any etiological relationship between Brucella infection and nummular keratitis. One of the commonest ocular findings in experimental brucellosis is a corneal infiltrate similar to that present in nummular keratitis. Further study of cases of nummular keratitis for the possibility of Brucella infection will be needed to elucidate this question.

In 1 patient in this series the uveitis had progressed to phthisis bulbi. The eye was painful and was enucleated. Histological examination showed a nonspecific uveitis with several conspicuous lymphoid nodules (fig. 7), a picture quite suggestive of that of periodic ophthalmia in horses.

None of these patients exhibited any symptoms of acute Brucella infection, and only 1 patient had a prolonged, low-grade fever. Most of the patients gave a history of a previous unexplained fever with malaise and various vague symptoms. In every case careful questioning brought out the history of ingestion of raw milk or raw milk products, and in several instances contact with animals with probable Brucella infection. Some of the patients were below par from a general physical standpoint but others were reasonably healthy and a few were actually robust.

The clinical diagnosis of brucellosis in these patients was of necessity based almost entirely on serologic and sensitivity evidence of the infection, although it was bolstered by a suggestive history. Brucella organisms were not cultured in any case, and unlike the cases reported by Green, these must all be considered to be in the chronic or possibly healed stage of the disease. The agglutinations, done against B. suis and B. abortus antigens of living organisms, were positive in dilutions of 1:40 to 1:320 in 12 of the 15 cases. One patient had an agglutination titer of only 1:20. In 1 other patient the agglutination reaction was negative, but the complement fixation reaction was strongly positive. In the last patient agglutination tests and the complement fixation reaction were both negative, but the cutaneous sensitivity test was violently positive and there were no other etiologic factors. Cutaneous sensitivity tests were not routinely done for the reason they appeared to disturb and invalidate later agglutination tests. They were done in five instances, being positive in 4 and negative in 1 patient. Brucella complement fixation tests were performed in 8 of these patients and were positive in 6.

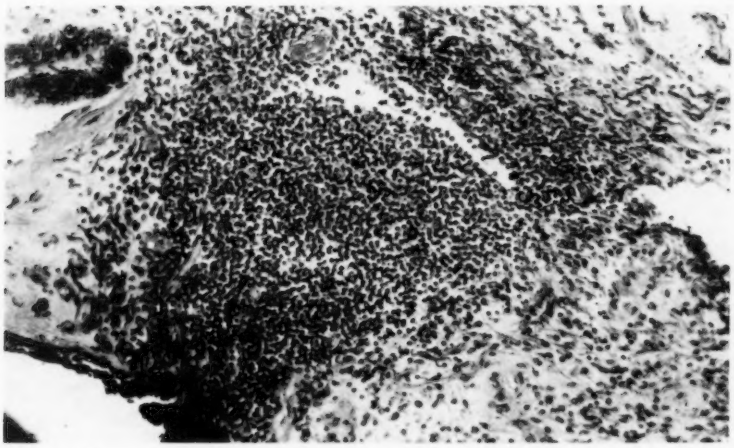

FIG. 7 — Microphotograph of brucellosis uveitis:
(a) Lymphoid nodule at root of iris.

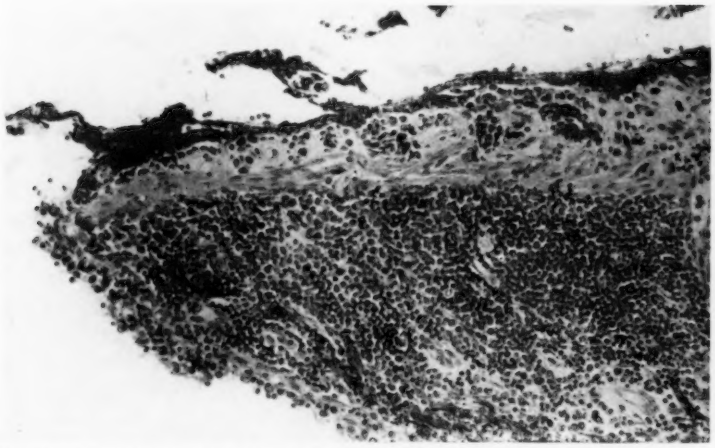

FIG. 7 — Microphotograph of brucellosis uveitis:
(b) Lymphoid foci near iris sphincter.

Positive serologic or sensitivity tests are, in the last analysis, only evidences that the patient has at one time in life had a Brucella infection, from which he may have recovered or may have passed into the chronic stage. Moreover, brucellosis is a relatively widespread disease. The question therefore naturally arises — what significance should be attached to these positive reactions?

In this entire series of patients, Brucella agglutinations were performed in 176 instances and were positive in 22, an incidence of 13 per cent. If the positive reactions are of diagnostic significance, and if brucellosis is an important factor in the causation of uveitis, the percentage incidence of positive agglutinations should be higher in a series of patients with uveitis than in a series of "normals," and should be somewhat comparable to the percentage incidences in medical patients in whom brucellosis was suspected as a factor responsible for their various symptoms. Unfortunately, we have no control series of nor-

mals for comparison of positive reactions; but we have a series of 47 medical patients in whom for various reasons chronic brucellosis was suspected and whose serums were sent to the Wilmer Institute for examination. Eight of these patients gave positive reactions, an incidence of 17 per cent. Thus, the incidence of positive agglutinations in the entire uveitis series is almost as high as in a group of medical patients suspected of having chronic brucellosis. This evidence suggests the incidence of brucellosis was higher than normal in the uveitis series, but is not statistical proof. Out of the 22 uveitis patients with positive Brucella agglutinations, only 8 patients (36 per cent) had definite evidence of some other factor which might be the cause of the uveitis. Out of the 154 patients with negative Brucella agglutinations, 72 patients (47 per cent) had evidence of some factor which appeared to be a definite cause of the uveitis. Thus, while the incidence of other definite factors is slightly lower in patients with positive Brucella agglutinations, the difference is not of statistical significance. It is therefore impossible from the evidence now available to obtain statistical proof that positive Brucella agglutinations are of diagnostic significance in establishing the etiology of uveitis. Such positive reactions are, however, suggestive evidence; and in general a positive Brucella agglutination is probably of about the same value in the diagnosis of Brucella uveitis as is moderate tuberculin hypersensitivity in the diagnosis of tuberculous uveitis.

A total of 76 complement fixation reactions for both Brucella and gonococci were performed on patients in this series. There were 21 positive reactions with Brucella, but 18 of these also showed a positive reaction to gonococci.

It is clear the diagnosis of chronic brucellosis as the etiologic factor in uveitis must always be presumptive, since the isolation of the actual organisms is impossible and there is no therapy so adequate we can apply a therapeutic test. However, despite the presumptive character of the diagnoses the accumulated evidence indicates brucellosis is probably an etiological factor in uveitis, and it may be quite an important one.

Since a diagnosis of chronic Brucella uveitis can never be made with certainty, the incidence shown in this study (7.5 per cent) represents a maximum. The actual incidence of Brucella uveitis may be anywhere below this figure.

SUMMARY

A series of 200 patients with endogenous uveitis examined in the Wilmer Institute between July 1, 1939 and July 1, 1943 is analyzed and the general problem of sarcoid and Brucella infections is discussed. No strictly out-patients are included in this series, the cases therefore comprising a group with uveitis more severe than average.

Each patient was subjected to an extensive diagnostic survey similar to that carried out in a series of 562 patients with uveitis previously reported. In addition, special tests for sarcoid and for brucellosis were performed in this series of patients. Such tests were done routinely whenever there was any suspicion of either of these diseases. Lymph gland biopsies for sarcoid were performed on 51 patients and serum albumin-globulin levels on 71 patients. Brucella agglutinations were done on 176 patients, Brucella complement fixation tests on 76 and Brucella opsonocytophagic indexes on a number.

In the etiologic classification, 15 cases of uveitis are diagnosed as definitely due to sarcoid. A histological diagnosis of sarcoid was made in each of these patients. In this group of patients it was noted that skin and bone lesions were notably scarce, that normal serum glob-

ulin levels were encountered much more frequently than has been reported in other series of sarcoid patients, and that lymph gland biopsy may yield evidence of this disease even though the lymph gland is not enlarged. Since strictly outpatients were excluded from this series, it is assumed that the actual incidence of sarcoid in uveitis is in the neighborhood of 3 per cent among all such patients seen in the Wilmer Institute.

Fifteen cases of uveitis are classified as "probably" due to brucellosis. The difficulty of diagnosing chronic brucellosis is pointed out, and the uncertainty of any diagnosis of Brucella uveitis is emphasized. The actual incidence of Brucella uveitis is assumed to be anywhere below the 7.5 figure reported in this series.

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DISCUSSION

S. JUDD BEACH, M.D., Portland, Maine: This outstanding report continues the classic contribution to the etiology of iritis presented by these authors two years ago before the Ophthalmological Section of the American Medical Association. Their position is judicial except perhaps with regard to focal infection. Before admitting the agency of foci they require that "complete diagnostic surveys have revealed no other etiological factor to which the uveitis may be logically attributed." This almost implies that (1), uveitis may be due to focal infection, yet (2), it cannot be so caused in patients who have tuberculosis, or syphilis, or similarly, brucellosis.

In contrast, take the case of a young woman, a librarian, who consulted us 15 years ago for recurrent iritis of the left eye. Investigation revealed only an extreme sensitivity to tuberculin. The disorder yielded to desensitization. Two years ago a quite different uveitis attacked the fellow eye, involving the deep corneal stroma. Tuberculin was ineffective. During further study, sulfonamides were administered with immediate relief. Furthermore, withdrawal because of leucopenia was followed by recurrence. Examination revealed 100 agglutinin titer to brucellosis, but she admitted tenderness in two teeth. Though roentgenologically negative, they showed abscesses on extraction. Immediate recovery followed.

Dr. Woods and Dr. Guyton are to be commended for insisting that foci of infection can be merely a coincidence in uveitis. Yet if foci are ever responsible, it is unsafe to hold them necessarily innocent in patients with evidence of tuberculosis and brucellosis.

This case suggests further study regarding response to sulfonamides as an indication of focal infection.

JACK S. GUYTON, M.D., Baltimore, Md.: I wish to thank Dr. Beach for his kind discussion. As he says, we have adopted a somewhat negativistic attitude toward foci of infection. This is perhaps a swinging of the pendulum a little too far in the other direction. In years gone by I think we searched for foci of infection and believed in them about as much at the Wilmer Institute as anywhere. Not only did we look for them in the case of uveitis, but we searched for them before even doing a cataract extraction or before doing a glaucoma operation.

The reason we began to swing the pendulum in the other direction was that when we made our first survey of uveitis patients we learned that the incidence of foci of infection in the group of uveitis patients as a whole was exactly the incidence in a good control series of patients without uveitis. Second, we found that the effect of removing, or partially removing, foci of infection did not decrease the incidence of recurrence of uveitis in our group of patients; therefore, we did adopt a somewhat negativistic attitude.

I might say that in our criteria for diagnosing uveitis as caused by a focus of infection, we did require that the patient not have evidence of any other factor which might be logically responsible for uveitis in order to make what we called a certain diagnosis of focus of infection as the cause; but in order to make a so-called presumptive diagnosis of focus of infection being the cause we did diagnose some cases as being due to foci of infection if it seemed more likely that the focus of infection was related to the uveitis than that some other factor was causally related.

I may supplement some of Dr. Woods' remarks by saying a little bit about the practical diagnosis of sarcoid. Sarcoid can be suspected from the ocular examination if there are large nodules in the iris. I mean very large nodules, and these nodules are somewhat juicier and pinker than those usually seen in tuberculosis and syphilis. About half the eyes that have sarcoid will show such nodules. Next an x-ray of the lungs is of considerable importance, because about 90 per cent of the patients with sarcoid show some infiltration in the lungs, usually near the bases or at the roots of the lungs. Third, obvious skin lesions may indicate sarcoid.

Fourth, an elevation of the serum globulin has been suggested in recent years as a good criterion for diagnosing sarcoid, and in various series of sarcoid reported in the medical literature, over 90 per cent of the patients with active sarcoid have shown an elevation

of the serum globulin to above 3 per cent. Unfortunately in our series 6 of the patients did not have an elevated serum globulin. Thus while an elevated globulin is good positive evidence, a normal globulin level is not good negative evidence. Furthermore, there are a few other conditions, such as active lymphogranuloma venereum, which can cause an elevated serum globulin.

Fifth, most patients with sarcoid show considerable anergy to tuberculin, that is, lack of sensitivity. They may fail to react to huge doses of even 10 milligrams intracutaneously, but this is not always so. It is sometimes difficult to distinguish between tuberculosis and sarcoid, because an occasional patient with sarcoid will react to .001 miligrams of tuberculin, and 3 of those in our series reacted to .01 milligrams of tuberculin.

Sixth, some of the patients with sarcoid show a definite eosinophilia, and in most of them the sedimentation rate is increased.

The final criterion for diagnosis of sarcoid must be histological demonstration of lesions. We biopsied 56 lymph glands out of these 200 patients and of the 15 patients who had sarcoid, only 3 of them had generalized enlargement of the lymph glands of the body. Three of them had absolutely no palpable lymph glands anywhere in the body, and we had to go in and find the lymph gland blindly, yet in all 3 of these there were definite histological evidences of sarcoid. In the remaining 9 there were a few palpable lymph nodes, and whenever there was one palpable we took that one out. We took out a great many normal lymph glands, because we biopsied 41 normal lymph glands and 15 sarcoid lymph glands.

It is not always pleasant to subject patients to biopsy of the lymph gland, but if they have other suggestive evidence of sarcoid I believe it is entirely justifiable to remove a lymph gland and make a positive histological diagnosis.

I am sorry no one criticized our presentation of brucellosis as an etiologic factor in uveitis. The diagnosis of brucellosis, as Dr. Woods said, is a presumptive one and we have no actual proof that brucellosis causes uveitis in humans, at least during the chronic stage. In experimental animals we know that 10 per cent of the guinea pigs inoculated systemically will develop an active keratouveitis. Furthermore, there is good evidence that most horses with recurrent endogenous uveitis, known as periodic ophthalmia, have brucellosis. As far as humans go we must remember that in certain sections of this country around 6 per cent of all the people are supposed to have had brucellosis at one time or another. In the literature there are literally hundreds of reported cases of human brucellosis in which the diagnosis has been proven; yet Dr. John Green in an exhaustive survey of the literature was able to find only seven instances of uveitis in proven cases of brucellosis, and it is possible that these may have represented coincidences. Furthermore, Rutherford examined 63 cases with proven brucellosis ophthalmoscopically and did not find uveitis in any of them. Therefore, most of our evidence that brucellosis may cause uveitis in humans is based on experimental work. This experimental evidence is solid, and we believe brucellosis does account for at least some cases of human uveitis.

As to the effect of treatment of brucellosis, we have not definitely benefited the patients with uveitis in whom we attributed the inflammation to brucellosis. There is not any treatment for chronic brucellosis which is so efficacious that we can apply a therapeutic test as to the cause of the uveitis.

THE DICKEY OPERATION FOR PTOSIS

Results in 21 Patients and 30 Lids

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and
ULRICH FRITSCHI, M.D.
SAN FRANCISCO, CALIFORNIA
BY INVITATION

IN 1935 Dickey conceived the idea of correcting ptosis, in those cases with normal function of the superior rectus muscle, by using a sling of fascia lata through the middle third of that muscle and attaching it to the tarsus. This was published in 1936[3] as a preliminary report. Although the operation has been in use at the University of California since that time no further cases were reported until Gifford and Puntenney[4] described their results in 5 cases in which they used a modification of the Dickey procedure.

Many operations have been devised for ptosis. They may be divided into two principal classes; one, to be employed in cases in which there is a paralysis or paresis of the superior rectus, and the other in those in which this muscle has normal function. The Dickey operation is included with the latter.

The Blascovics[2] operation has been used rather widely. In this operation the upper portion of the tarsus is removed and the levator which has been resected from it is brought forward and attached to the remaining portion. For correction of complete ptosis the results of this operation have been disappointing.

In 1897 Parinaud[7] and Motais[6] conceived the idea of substituting the middle third of a normal functioning superior rectus for that of the paretic levator. Since that time the basic principle of the Motais operation has been almost universally used for ptosis in the presence of a normal functioning superior rectus muscle. The operation was modified by Shoemaker[8] in 1907 by making an external incision which gave a better exposure and permitted firmer suturing of the muscle tongue to the tarsus. Kirby[5] also made some minor changes in the operation.

There are several disadvantages to the original Motais and its modified forms. The strip of superior rectus sutured to tarsus may tear out. Even though Kirby[5] advised that this be immediately reattached by a second operation, this complication is a risk to be avoided if possible. Another disadvantage is the fact that, due to the small strip being attached at one point only, there is a tendency to "tenting" or inverted V formation of the upper lid. The difficulty of doing this operation in small children is such that, in discussing the Motais operation, Bielschowsky[1] stated: "It is not advisable to operate on young children because a child's superior rectus muscle is so small and delicate the sutures may easily tear through." The Motais also does not permit an adjustment of the degree of correction to make the lid conform to the position of the lid on the other side. The superior rectus muscle is disturbed

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and injured and its normal attachment altered. Furthermore the dissection entailed is considered difficult. Finally, according to Wheeler,[12] rarely, near the attachment of the superior rectus muscle to the upper lid, entropion may occur and cilia rub on the cornea. Because of these disadvantages various methods of employing the intact superior rectus have been devised.

Wiener[13] in 1928 advocated the operation suggested by Cannos and Arrangues in which the levator is severed almost 6 mm. from its tarsal attachment and sutured to the tendon of the superior rectus, thus attaching the tarsus to the superior rectus by means of a portion of the levator tendon. As Gifford and Puntenney[4] point out this would "seem to produce an adhesion to the muscle too far from the lid border to accomplish the desired elevation of the lid in cases of complete ptosis."

In 1935 Trainor[11] presented an operation in which a strip of the upper edge of the tarsus is passed under the superior rectus muscle. With the lid everted an incision is made through the tarsus 2 mm. from its superior edge and parallel to it for a distance of 7 to 10 mm. This strip of tarsus with the conjunctiva adherent is then passed beneath the superior rectus tendon and sutured back in place. The operation is described and illustrated in detail in the Wiener and Alvis book.[14] These authors recommend it as the simplest operation for ptosis. The operation does not, however, allow for an adjustment of position of the lid to balance the other side. It has disadvantages pointed out by Spaeth[9] and Gifford and Puntenney[4] in that it forms an undesirable adhesion in the upper cul-de-sac which interferes with the movement of the eye and that it buries an epithelial surface (the conjunctiva covering the strip of tarsus) beneath the superior rectus muscle.

In 1938 Wheeler[12] advocated correction of ptosis by attachment of strips of orbicularis muscle to the superior rectus muscle. Through an external incision the dissection is carried through the orbicularis muscle, the tarso-orbital fascia, the levator muscle and Tenon's capsule, and the superior rectus picked up on a hook. Strips of orbicularis are then dissected up from the tarsus and the ends towards the canthi which have been cut, are attached to the superior rectus muscle with fine catgut. This operation apparently does away with many objections to the Motais, but as Wheeler himself pointed out, the dissection might be considered difficult.

Realizing that there is no perfect substitute for a paralyzed muscle we feel, after eight years of experience with it, that the Dickey operation has been entirely satisfactory and that it does not give rise to most of the objections to other operations. Spaeth[10] in commenting on the Dickey operation says it undoubtedly provides the firmest attachment that can be obtained between the superior rectus muscle and the tarsus.

TECHNIC OF THE DICKEY OPERATION

General anesthesia has been employed in all the cases. While the preliminary part of the operation on the lid is being done a strip of fascia lata 8 to 10 cm. long and 8 mm. wide is obtained by a general or plastic surgeon. The technic employed in obtaining the fascia is to make a 2 cm. incision on the midlateral aspect of the thigh at the junction of the upper and middle third. This incision is carried down through the subcutaneous tissue until the fascia lata is exposed. The fascia is bluntly separated from the overlying subcutaneous tissue toward the knee. Two short parallel incisions about 8 mm. apart are made through the fascia in line of its fibers and the fascia between them divided. This free end of the fascial strip

is passed through the loop of a fascial stripper and held taut by a heavy clamp. The stripper is forced down the thigh to the knee, where the fascial strip is cut across by the blade of the stripper. The subcutaneous tissue and skin are closed in layers by interrupted sutures. The fascia lata is folded with the muscular surfaces together. This gives it additional strength and also guards against cutting through by the sutures.

While the fascia is being obtained a conjunctival incision is made over the superior rectus muscle, the muscle exposed and picked up on a hook (fig. 1-A). The central third of the muscle is picked up and separated back a short distance. The folded fascia lata is now passed beneath the central third of the muscle (fig. 1-B). The conjunctiva and Tenon's capsule are sutured with No. 0000 catgut over the lateral portions of the muscle. An additional conjunctival suture is placed over the central third of the muscle between the two folded bands of fascia, so that no raw surfaces

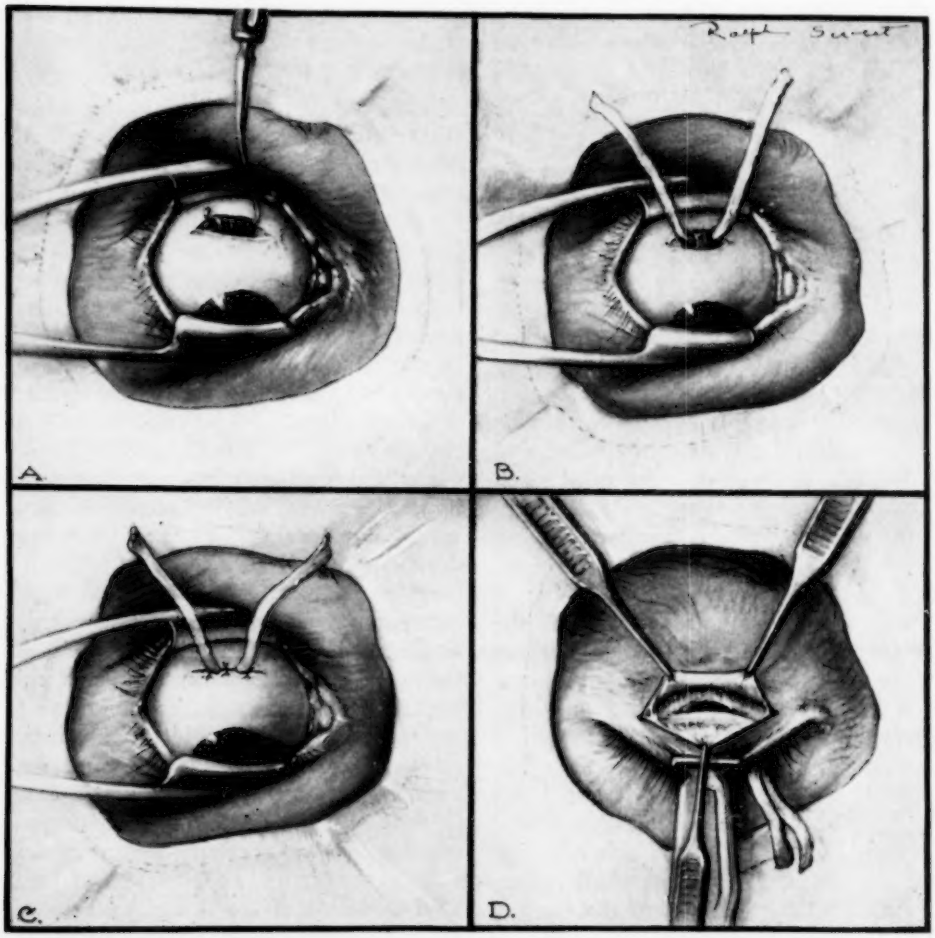

FIG. 1 (Cordes and Fritschi) — A, Isolation of superior rectus muscle; B, fascia lata strip under central third of tendon; C, conjunctiva and capsule sutured; D, tarsus exposed, showing 5 mm. incision made through levator tendon at upper tarsal edge.

remain. This does away with the possibility of conjunctival adhesions (fig. 1-C). The lid speculum is removed.

A lid clamp with a conjunctival plate (or a Jagers lid spatula) is introduced under the lid. A horizontal incision is made through the skin and orbicularis 8 mm. from the lid margin and about 20 mm. in length. The dissection is extended through the orbicularis muscle until the tarsus is exposed well down to the lid margin and upward to the upper edge of the tarsus and the levator tendon.

At the midpoint of the upper edge of the exposed tarsus a 5 mm. incision is made through the levator tendon into the conjunctival sac (fig. 1-D). The fascia lata strips are now brought through this opening by forceps and pulled down over the tarsus (fig. 2-A).

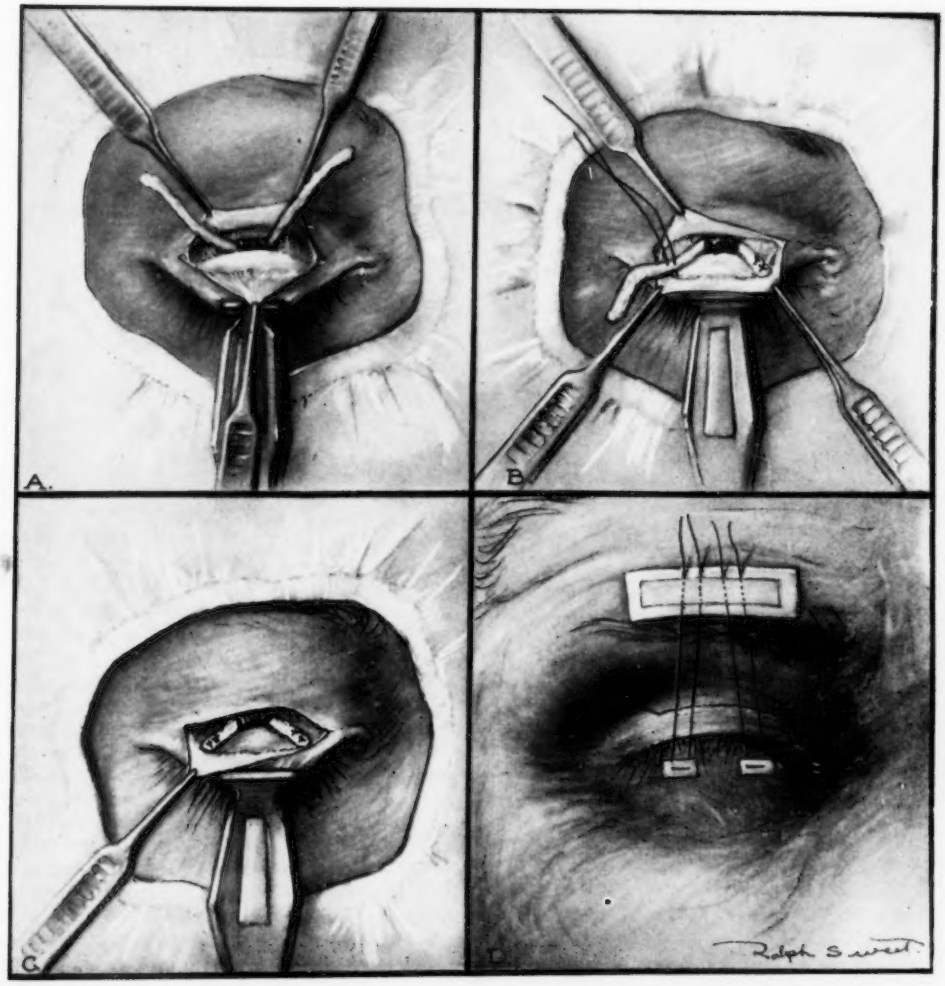

FIG. 2 (Cordes and Fritschi) — A, Fascia brought through incision in levator; B, sutures through tarsus and fascia, showing outer sutures tied; C, fascia lata sutured to tarsus; D, lid sutures as advocated by Gifford and Puntenney.

A double armed No. 0.009 silk or linen suture is placed in the tarsus at the junction of the outer and middle third and about 4 mm. from the lid margin. Both needles are then passed through the folded fascia lata, tied firmly and the suture cut. A second firm bite is taken in the tarsus with another double armed suture, just below the site of the first suture, the suture is passed through the fascia lata, tied and cut (fig. 2-B). This reinforces the first suture. Another double armed suture is similarly placed at the junction of the middle and inner third of the lid and about 4 mm. from the margin (fig. 2-B). The lid clamp is removed and by traction on the fascia loop or sling the site of this suture is determined. The point chosen for this mattress suture will determine the effect obtained and therefore should be carefully estimated. Originally Dickey advised that the point should be chosen that will lift the lid margin to a point 3 mm. below the limbus. More recently we have chosen a point 2 mm. from the limbus in most cases, while Gifford and Puntenney found that they obtained satisfactory results with the lid margin 1 mm. from the limbus. The suture is now put through the fascia at the chosen point and the suture tied lightly. The position is inspected and if it is satisfactory the sutured is tied tightly. Another mattress suture is placed just below this suture, introduced through the fascia and tied. The sutures are cut and the excess fascia lata trimmed off (fig. 2-C).

The position of the lid is now inspected to be certain that the lid margin is horizontal. If one side is higher than the other the fascia is adjusted where it passes through the middle third of the superior rectus. In 1 case (case 19) with partial ptosis where the outer half of the lid drooped more than the inner half the fascia was adjusted to compensate for this.

The skin incision is closed with a continuous subcuticular suture, the fascia sutures being buried. The fibers of the orbicularis fall together of their own accord.

In the past the cornea was protected by the use of ointment or a moist chamber. We feel the method employed by Gifford and Puntenney (based on Friedenwald's procedure) is probably better and we are now using it. By this method the "protection of the cornea is secured by passing two double armed sutures through the lower lid and out through the lid border. The sutures are attached to the brow on adhesive tape so as to pull the lower lid up to the new level of the upper lid" (fig. 2-D).

The subcuticular suture is removed on the sixth day. The sutures of the lower lid which may be relaxed by removal of the tape for dressings renewed every second day, are also removed on the sixth day.

CASE REPORTS

The following case reports from the University of California show the results obtained with the Dickey operation for ptosis. In this series 21 patients and 30 lids were operated upon by various members of the staff.

Case 1. G. C., a boy of 20 months, was brought to the Clinic with the history of bilateral ptosis since birth. The drooping was so marked that he was compelled to throw his head back in order to see. The right side was operated upon March 11, 1935, and at this time sufficient fascia was obtained for the other side. This fascia was preserved in alcohol and used on the left eye when it was operated upon six weeks later. The result obtained in the right eye was excellent while the operation on the left was a complete failure, the fascia, as found at re-operation, being entirely absorbed. Six months later the left eye was reoperated without difficulty and an excellent result obtained. This was Dickey's first case and since that time no further attempt has been made to use preserved fascia.

Case 2. Louis V., a boy of 4 years, was brought to the Clinic August 19, 1935, because of his inability to open his eyes. Aside from bilateral congenital ptosis the eye examination

was negative. On August 26, 1935 both lids were operated upon and excellent results obtained (fig. 3).

Case 3. M. R., a boy of 2½ years, was seen in January, 1936 because of a bilateral congenital ptosis. On February 4, 1936 both lids were operated upon with an excellent result. There were a few areas of corneal staining for a short time after operation but these cleared without corneal scarring (fig. 4).

Case 4. D. T., a boy of 9, reported with a bilateral ptosis which was almost complete in the left eye. The right lid was operated upon June 9, 1936 and the left lid August 13, 1937. A good result was obtained with the position of the lids in both eyes being 1 mm. above the upper pupillary border.

Case 5. H.L., a woman 20 years old, gave the history of progressive ptosis of both lids over a five year period. The ptosis had apparently been stationary for the preceding year. After a medical examination which was essentially negative the left lid was operated upon March 23, 1937 with a good result. Patient had a vertical diplopia that persisted until the right side was operated upon on October 27, 1937. An excellent result was obtained, with good lid folds in both eyes.

Case 6. D. T., a boy of 5, reported with a bilateral congenital ptosis more marked on the right side. The right lid was operated upon June 18, 1937 and the left on August 13, 1937 with excellent results.

Case 7. C. H., a boy of 15, showed a congenital ptosis of the left side and was operated upon June 4, 1937. Following surgery there was an overcorrection of about 1 mm. Patient had diplopia on extreme elevation. Patient was so well satisfied that he would not consent to further surgery.

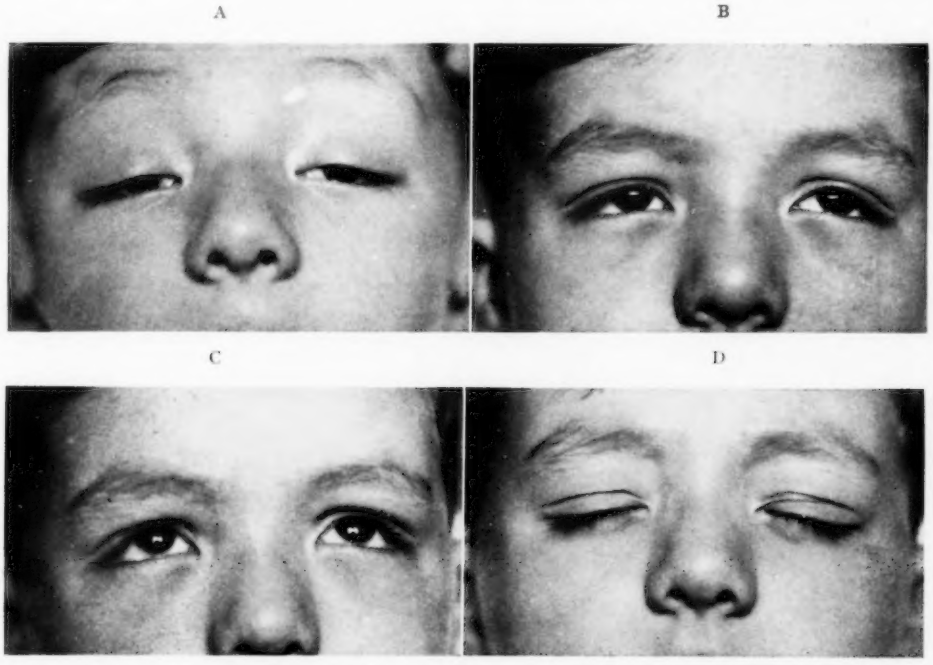

FIG. 3 (Cordes and Fritschi) — Case 2. L. V., age 4, bilateral congenital ptosis. A, before operation; B, after operation. Note lid folds; C, position of lids in elevation; D, lids closed.

Case 8. B. L., a man of 21, had bilateral ptosis and epicanthus which had been operated elsewhere two years before entry. A modified Motais had been done on both sides. The patient stated that the condition was worse following surgery. In spite of a slight weakness of the left superior rectus it was decided to do a bilateral Dickey operation. Both sides were operated upon on June 15, 1938. At the time of surgery there was scar tissue present over the superior rectus and it appeared as

though the tongue used in the Motais had retracted. Little difficulty was encountered during the operation and patient obtained an excellent cosmetic result. He had occasional diplopia during the year and a half he was under observation. This however was not troublesome enough to make him consent to further surgery.

Case 9. A. S., a boy of 9, was operated upon August 7, 1937 for bilateral congenital ptosis with satisfactory results.

Case 12. B. M. A., a boy of 16, had a congenital ptosis of the right side. His grandfather had a similar condition. The right side was operated upon on January 31, 1938 and an excellent result obtained. Seven months later he returned with a definite gray band across the cornea and running through the lower third of the pupil. The lids were sutured together for 3 weeks and at the end of this time there was no staining. When seen six months later the eye was quiet but had

FIG. 4 (Cordes and Fritschi) — Case 3. M.R., age 2½, bilateral congenital ptosis. A and B, before operation; C, six weeks after surgery; D, three years after operation.

Case 10. T. R., a boy of 4, was operated upon September 3, 1937 for a complete congenital ptosis of the left side. The result was excellent. A year later the mother stated that the psychological effect on the child was "amazing."

Case 11. S. J., a female child of 13 months, had a right sided congenital ptosis which was operated upon October 28, 1937. The result was entirely satisfactory. This is the youngest case that has been operated upon. There were no particular difficulties involved due to the small size of the patient.

a fine linear scar across the lower third of the pupil.

Case 13. F. C., a boy of 9, had a left sided partial congenital ptosis. The lid margin was at the center of the pupil. Operation was performed January 4, 1938, the lid margin being placed at the same level as that of the fellow eye, with excellent result. The patient had diplopia on extreme elevation which was not very troublesome but was still present eighteen months later.

Case 14. H. G., a boy of 9, had a right sided congenital ptosis that was operated upon February 20, 1940 with a satisfactory result.

Case 15. N. G., a young woman of 23, had a complete ptosis of the left side associated with an internal strabismus of 25 degrees arc. There was a right hyperphoria together with some weakness of the left superior rectus. The left eye was amblyopic. Before coming to the Clinic the patient had had an operation for strabismus and two operations for ptosis. As well as could be determined she had had a shortening of the superior rectus with attachment of the levator for her first ptosis operation. The second operation for ptosis was a Motais. Both had been failures so that when we first saw her she had a complete ptosis of the left side (fig. 5-A). It was decided that a Dickey operation would offer the best cosmetic results even though there was some weakness of the superior rectus together with a hypophoria. Because the eye was amblyopic there was no fear of diplopia.

The Dickey operation was performed on July 2, 1940. Because of the scar tissue from previous operations the dissection was a little more tedious but not difficult. April 29, 1941 she was reoperated for strabismus. Cosmetically the result was extremely satisfactory particularly in view of the failure of the two previous operations. In the primary position there is a little hypophoria, a little drooping of the lid and some fullness at the outer corner (fig. 5-B). While the upward motion of the left eye is somewhat limited due to weakness of the superior rectus the lid follows the eyeball upward so that there is no great cosmetic disfigurement (fig. 5-C). As the left eye is amblyopic, there has been no diplopia. While the end result is not as satisfactory because of existing circumstances as in the uncomplicated cases we feel that the operation can be classed as successful.

Case 16. D. C., a boy of 4, had bilateral congenital ptosis for which both sides were operated. The results were satisfactory although the left lid was a mm. lower than the right due to a slight undercorrection.

FIG. 5 (Cordes and Fritschi) — Case 15. N. G., age 23. Congenital ptosis; internal strabismus L. E. 25° arc, some weakness of left superior rectus, amblyopia left eye; two previous operations for ptosis, probably levator shortening and Motais. A, after two previous operations; B, after Dickey operation; C, position of lid in elevation. No diplopia because of amblyopia and suppression.

Case 17. T. P., a 3 year old male, had a right sided complete congenital ptosis that was operated upon February 7, 1941 with satisfactory results. In postoperative care some difficulty was encountered in trying to prevent the cornea from drying out and in spite of all efforts a small corneal infiltrate and resultant scar developed.

Case 18. L. K., a young man of 17, had bilateral congenital ptosis. The right side was

operated upon June 11, 1941 and the left on May 1, 1942 with excellent results in both.

Case 19. C. C. M., a boy of 8, had partial ptosis on the right side with the lid margin at the level of the middle of the pupil. The outer part of the lid was considerably lower than the inner half. The lid was operated upon May 26, 1942, particular care being taken to see that the outer and inner halves of the lid were on the same level. The result was excellent, the position of lid being on the same level as that of the other eye (fig. 6).

COMMENTS

This series consists of 21 patients on whom ptosis was corrected by the Dickey operation in 30 lids. Of this group 18 were males and 3 females. The youngest to be operated (case 11) was a female child of 13 months. In view of the difficulty of doing a Motais at this age, it is interesting to note that no complications were encountered and that.

FIG. 6 (Cordes and Fritschi) — Case 19. Calvin M., age 8. Right sided partial ptosis from birth. Dickey operation right eye. A, before operation; B, after surgery; slight over-correction of inner half emphasizes necessity of careful adjustment of fascia.

FIG. 7 (Cordes and Fritschi) — Case 21. B. F., age 10. Left sided congenital ptosis. A, before surgery; B, after Dickey operation.

Case 20. F. M., a boy of 8, had either congenital or obstetrical ptosis of the right side which was operated upon July 18, 1942 with excellent results.

Case 21. B. F., a boy of 10, had left sided congenital ptosis (fig. 7-A). A Dickey operation was done August 4, 1942 with a satisfactory result, the lid now being at the same level as the right (fig. 7-B).

the result was excellent. The oldest patient was a woman of 23 (case 15). Her case warrants special comment which will be made later.

In this series there was 1 outright failure (case 1), probably due to the fact that preserved fascia was used. In 2 instances (case 15, case 16) there

was a little undercorrection. In case 15 where the lid had been unsuccessfully operated upon twice there was a good deal of scar tissue which made the dissection a little more difficult; in addition a fullness of the outer half of the lid tended to emphasize this undercorrection (fig. 5-B). In one instance (case 7) there was an overcorrection of about 1 mm.

Three cases (case 1, case 8, and case 15) had had previous unsuccessful surgery for their ptosis. In 2 instances the Motais had been used. Aside from the little inconvenience of dissecting through scar tissue no difficulties were encountered re-operating these cases, and good results were obtained in all of them.

Three of the patients had corneal involvement following surgery, 2 of them having a small resultant corneal scar. This occurred during the early convalescent period. This percentage seems rather high and we feel that the use of the 2 double armed sutures through the lower lid as suggested by Gifford and Puntenney[4] and which we are now using will very materially reduce the occurrence of this complication.

It is noteworthy that in none of the cases was there any tendency to "tenting" or inverted V formation of the palpebral fissure. In addition in almost every case the lid folds had a normal configuration (figs. 3, 4, 6, 7).

There were 4 cases of diplopia. In case 5 this disappeared after the second eye had been operated upon. In cases 7 and 13 diplopia appeared only in extreme elevation. This was not sufficient to be troublesome and annoyed the patient far less than the ptosis. In case 8 a very slight weakness of the superior rectus, diagnosed preoperatively, accounted for the diplopia which was not sufficient to be very annoying. In case 15 there was also a slight weakness of the superior rectus. An amblyopic eye with suppression prevented diplopia. Case 8 emphasizes the necessity of careful study of the superior rectus and the eye before operation is contemplated.

Slight weakness of the superior rectus is no contraindication to the Dickey operation if there is amblyopia or suppression. Also if the paresis is so mild as to cause diplopia only on extreme elevation the Dickey procedure is preferred to an operation in which the lid is fixed and does not follow the movement of the eyeball. In the Dickey operation the lid is attached by the fascia sling to the tendon insertion of the muscle so that the entire superior rectus aids in holding up the lid, consequently the presence of slight weakness does not materially alter the action of the muscle. In the Motais in which the effect is produced by only a portion of the muscle any weakness would be exaggerated.

In a case done elsewhere diplopia was so marked because of paresis of the superior rectus that it became necessary to undo the operation and restore the ptosis. It is noteworthy that this is a very simple procedure. Reaching into the upper conjunctival sac with a small muscle hook such as that used in the O'Connor operation the fascia strips are easily picked up on either side and cut with tenotomy scissors, thus restoring the former ptosis.

The fact that obtaining the fascia lata necessitates an additional surgical procedure has brought forth some criticism. Ptosis is such a disfiguring and distressing condition that any aid obtainable in overcoming this disfigurement seems justifiable. The obtaining of the fascia is not difficult and the scar on the thigh is a small one. In none of the cases in which the fascia was obtained was there any postoperative complication contingent upon its removal. In view of the many advantages of the Dickey operation we do not feel that the objection to the additional surgical

procedure required in obtaining the fascia is justified.

Gifford and Puntenney[4] have modified the Dickey operation in that they operate entirely through a cutaneous incision and pass the fascia sling under the entire superior rectus muscle. We can see no real disadvantage to these procedures. We have felt that for the average operator there was less difficulty in isolating the superior rectus through the conjunctival incision. After the fascia has been put through the central third of the muscle the conjunctiva is closed on either side right up to the fascia. In addition another suture brings the conjunctiva together between the two fascia bands (fig. 1-C). The conjunctiva is closed with No. 0000 catgut so that it is unnecessary to remove sutures. To date we have seen no adhesions form in the conjunctival sac and in one case previously noted cutting the fascia bands has restored the ptosis. Gifford and Puntenney have apparently limited the Dickey operation to cases of complete ptosis. We have used the operation with excellent results in those cases of partial ptosis in which an attempt is made to elevate the lid to the level of the fellow eye (cases 13 and 19) (fig. 6). In these cases particularly, it seems advisable not to destroy what action the levator may have. Consequently it seems to be an advantage to make only a small opening through the tendon at the upper tarsal edge through which the fascia bands can be passed (fig. 1-D), rather than completely sever the tendon by using a cutaneous incision for the entire operation. Thus in the event of a failure of the operation the partial ptosis would be no worse than it was preoperatively. In case 8 a Motais had been done elsewhere for partial ptosis. As well as could be determined it was done entirely through a skin incision. The result of the operation was a failure and the patient stated that the ptosis was worse than it had been before surgery.

We have felt that putting the fascial sling through the central third of the muscle allowed for a little better adjustment especially in cases such as No. 20 where one lid was lower than the other. It would seem that in these cases there would be less tendency for the fascia to slip than when it goes beneath the entire muscle.

We also have found that placing the lid border 3 mm. below the limbus is a little low and have been placing it at 2 mm. from the limbus. The final decision as to where the lid border will be placed, to a certain extent, depends upon the position of the lid in the normal eye. It should be emphasized that the position of the lid in the normal eye should be carefully studied before surgery is attempted.

SUMMARY

1. The Dickey operation for ptosis has been used at the University of California for eight years. During this time 30 lids have been operated upon on 21 patients with satisfactory results.
2. The operation can be used in small children, the earliest case in our series was a 13 months old child.
3. In only 3 cases was the operation followed by permanent diplopia; of these, 2 only on extreme elevation; the third only at times.
4. Three lids previously unsuccessfully operated upon by other methods were corrected without great difficulty.
5. In addition to its use in the presence of a normally functioning superior rectus the operation can be used to advantage in those cases of slight weakness of the superior rectus where that eye is amblyopic or suppressed. In these cases the result is better cosmetically than where the

position of the lid remains fixed and with amblyopia or suppression there is no fear of troublesome diplopia.

6. Due to the two point suspension there is no tendency for the inverted V formation of the lid and the skin folds show the normal conformation.
7. The operation permits adjusting the position of the lid to conform with that of the other side.
8. The operation has been found to be satisfactory in bilateral complete ptosis; unilateral complete ptosis and partial ptosis in those cases where the action of the superior rectus is normal.

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DISCUSSION

SANFORD R. GIFFORD, M.D., Chicago, Illinois: It is a pleasure to discuss this excellent paper by Dr. Cordes and Dr. Fritschi. They are advocating what I certainly agree is a fine operation. I agree with Spaeth that it produces the firmest attachment which can be obtained between the superior rectus and the tarsus and would go further to say that it is the best operation yet devised for the correction of complete ptosis associated with a normal superior rectus. By complete ptosis is meant ptosis in which the lid does not rise at all when the patient looks up, without an effort of the frontalis muscle, i.e., in which no action of the levator can be demonstrated. The modification made in the technic by Puntenney and myself is not instrinsically important and certainly does not detract a particle of value from the original conception of Dickey. Anyone can modify a procedure but how few of us have ever had a really useful original idea? Our idea in approaching the superior rectus through the skin was to simplify the procedure by avoiding the cut through the conjunctiva and to obviate the possibly undesirable effects of making a communication between Tenon's space and the conjunctival sac. It is evident from the authors' results in 21 cases that no real objection can be made to the operation on the latter score, since they observed no difficulties due to such a communication, which was closed off by careful conjunctival suturing. I do feel however, that the approach through the skin is simpler. The only difficulty, in one's first operations is to realize how far back the superior rectus really is when approached by this method and that it can only be exposed when the eye is held down with forceps. It is convenient to place a tension suture through the conjunctiva in the upper fornix, which holds this tissue away during the dissection and prevents it from being button-holed. With this method only five sutures are employed, those which hold the fascia to the tarsus, just as has been described and that which closes the skin. We do not insist upon the value of using the whole superior rectus

instead of its middle third except, again, that it seems simpler and gives a feeling of more security. The result is a little better on the right side in our first case in which the fascia was passed beneath the middle third in the left eye and beneath the whole muscle in the right which seemed to show a lack of tone when exposed at operation.

My only difference of opinion with the authors is that I still prefer shortening the levator by the Blascovics method in partial ptosis with some action of the levator. It is really not a much simpler procedure, but does obviate the second procedure of obtaining the fascia. My real reason for perferring it in such cases is that it gives, in most cases, as good a cosmetic result as the Dickey procedure and that the function of the muscles more nearly approaches the normal. It is usual, after the Dickey procedure, to have the lids remain slightly open when the eyes are turned up in sleep, though the cornea is not exposed, providing a normal Bell's phenomenon is present. The Blascovics procedure usually avoids this, and does avoid the occasional complication of vertical muscle imbalance. There have been some partial failures after the Blascovics, even when action of the levator seemed to be present and we now have a tendency to prefer the Dickey procedure when there is any doubt whether the levator action is sufficient to give a good result with the Blascovics. In complete ptosis there is, of course, no objection to approaching the superior rectus through the skin.

Since our paper, we unfortunately stopped taking photographs, but have performed the operation on 9 eyes of 8 patients in addition to the 5 cases then reported. The results have been really satisfactory in all cases. In the unilateral cases the fascial sutures are adjusted to raise the lid to the same level in the cornea as that of the other lid in its habitual position and this position has been almost exactly maintained in all the cases, except those to be mentioned. In one bilateral case an over-effect was obtained on one eye with a peculiar chemosis of the conjunctiva which protruded through the lids. It was necessary to excise part of this conjunctiva and to make an intermarginal adhesion to prevent recurrence of the chemosis. The lids were left closed by this adhesion for two months. On opening them the chemosis had disappeared and the position of the lid was satisfactory, practically identical with that of the other side. In one case previously reported a partial effect only was secured, requiring a later Machek procedure, which gave a good result.

No patient has complained of diplopia although in 1 case mentioned in our previous report a pre-existing hypotropia was made somewhat worse by the Dickey procedure. This was chosen because a previous Blascovics had failed. In no other case have we deliberately used the procedure in the presence of a known weakness of the superior rectus. The Machek operation, which I formerly employed in all types of ptosis, is now reserved for cases with complete ptosis associated with faulty action of the superior rectus. It is perhaps the simplest effective operation for that condition but one must be careful not to raise the lid too high as the cornea is especially apt to suffer from exposure when the superior rectus does not raise it up during sleep. When a partial but insufficient effect has been secured by a Blascovics or Dickey operation I have employed the Machek procedure to improve it, or a somewhat similar procedure which I hope to describe elsewhere. In complete failure of the Blascovics, one cannot repeat it but may do a Dickey or a Machek. If the Dickey fails completely one may, according to the authors, repeat it with prospects of a good result.

In any ptosis operation there can be no doubt that the best protection of the cornea is afforded by pulling up the lower lid with a Friedenwald suture. We have seen no corneal lesions since this has been done.

I am exceedingly glad that Dr. Cordes and Dr. Fritschi have brought this operation again to our attention and hope that many Academy Fellows will try it and will enjoy using it as much as we have.

C. A. DICKEY, M.D., San Francisco, California (by invitation): I have been associated with Dr. Cordes at the University of California Hospital for the past eleven or twelve years; and we have discussed this procedure at length, so there is very little that I can add. One suggestion that I might make is that if you decide to use this operation in a case of monocular partial ptosis, you must be careful to recognize the normal position of the non-ptotic lid. These patients with monocular partial ptosis utilize their frontalis to elevate the ptotic lid, and consequently there is a slight elevation of the non-ptotic lid. About two months ago ,I performed this operation on this type of case, and I matched the position of the lids. After surgery I noticed that the position of the non-ptotic lid was lower than before the operation. As a result of this, I may have obtained an overcorrection. In the future I think I will patch

the ptotic eye until relaxation of the frontalis muscle is obtained, thus permitting the nonptotic lid to assume its normal position.

Dr. Cordes mentioned a slight notching at the inner side of the lid margin in one of his cases; this, I feel, is due to an inequality of the fascial sling. Therefore it is well to be sure that the fascial strips are of equal length before closing the skin.

In the original procedure I placed the sutures down toward the lid margin, but I now feel that a better result is obtained if they are placed near the upper border of the tarsus.

W. B. LANCASTER, M.D., Boston, Mass.: I should like to call attention to a paper on ptosis that was published, I think, in 1918. The paper was delivered before this society. It was an attempt to evaluate at that period the different operations and two new operations were described. One was the first use of faschia lata in operations for ptosis and the other was an operation for advancement of the levator which was similar to the one that Blascovics published about seven or eight years later, the difference being that Blascovics goes in through the conjunctiva and that approach is not very easy except for a man who has done it a great many times. You cannot see very well what you are doing.

In this paper the approach was double, partly through the conjunctiva but also through the skin where you could see very well what you were doing. It is interesting that that paper was presented at about the stage of the first World War and this paper is presented at this stage of the second World War.

FREDERICK C. CORDES, M.D., San Francisco, California: I wish to thank the discussers. Just one more point that I want to bring out is that the edema which is present in the upper lid sometimes takes several months to subside. I also want to reply to Dr. Dickey that I have been after him for three years to publish further results on his operation and he was too modest to do so. That is why I gathered together all the cases that were done by the members of the staff and presented them again, because I feel that it is, at least in my hands, the best we have for ptosis.

Conservation of Hearing

TREATMENT OF IMPAIRED HEARING. WHAT CAN BE ACCOMPLISHED BY CONVENTIONAL PROCEDURES

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Conventional means usual or customary. Hence this limits my discussion to those therapeutic measures which are in general use among otologists in dealing with impaired hearing and eliminates those which have not, as yet, received general acceptance or employment. It calls for a frank and dispassionate appraisal of results by which we may evaluate the relative effectiveness of newer methods, the reports of which are to follow. While the use of hearing aids and specialized educational methods are certainly conventional measures, I shall omit more than casual reference to them from this discussion. This phase of the subject will be taken up by Dr. Reger and Dr. Hughson.

In this discussion we will assume adequate knowledge and skill on the part of the otologist, and correct performance of whatever procedure may be carried out. No method of treatment, old or new, can be fairly evaluated on any other basis.

Deafness is a symptom of some condition involving the conducting mechanism of the ear or the auditory nerve or both. If that condition is possible of reversal by our conventional methods of therapy, the results should be satisfactory. The simplest example of this would be some obstruction of the external auditory meatus, such as by cerumen. Another would be impaired hearing due to acute suppuration in the middle ear or mastoid. Adequate measures aimed at relieving the condition will allow restoration of hearing, depending somewhat upon how early treatment is instituted and how promptly the condition responds to it.

In the treatment of chronic suppurative otitis media the chief objective of the otologist will continue to be the arrest of the process and the healing of the ear, despite the not so uncommon observation that a patient will sometimes hear better when the ear is moist. Hearing aids of various kinds seem about as satisfactory as anything that can be offered to improve hearing, once the suppurative process is controlled. The paper drum of Blake which covers a perforation of the membrana tympani is often of material benefit to a patient's hearing. An old and often effective procedure, in cases in which the drum membrane has been destroyed and the ossicular chain necrosed, has been the use of simple prostheses of cotton or wool. Lillie and Kranz[5] found that this increased the hearing acuity for low frequencies by 10 decibels and for high frequencies by 30 decibels in a patient who had had a bilateral radical mastoidectomy. Hughson[4] employs cotton packs soaked in zepherin which he applies to the round window, stating that acuity is increased by immobilizing Scarpa's membrane. Pohlman[6] disagrees with this, feeling that the pack itself acts as an inefficient transformer. He has devised an ingenious diaphragm-rod prosthesis by which he can improve the acuity by as much as 30 decibels. The effectiveness of any prosthesis depends largely upon its proper placement and most often the patient himself can accomplish this better than can the otologist.

While acute catarrhal otitis media is generally a self-limited disease, residua from this condition, especially if recurrent, may result in later impairment of hearing. A recent excellent paper on this subject by Hoople and Blaisdell[3] suggests the importance of classifying these cases on an etiological basis. Treatment of the underlying cause, whether infection in the nasopharynx, pharynx or sinuses, or some non-infectious condition, such as allergy or endocrine imbalance, by evacuation of fluid from the middle ear, when present, either by paracentesis or inflation, may produce early restoration of function and may prevent later hearing loss.

Perhaps our greatest concern is with that large group of cases, usually rather loosely designated as conduction, or mixed type hearing impairment. The commonest cause of conduction deafness, especially in children, is obstruction of the eustachian tube from nasopharyngeal lymphoid

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tissue. These cases first manifest a drop in the upper tonal range, as shown by Crowe,[1] and early recognition and relief is important in preventing later hearing impairment. Of late some dissatisfaction has been expressed as to the adequacy of adenoidectomy in meeting this situation. Is this due to lack of surgical thoroughness, or is recurrence of lymphoid tissue a normal or usual event? From observations made on a large series of cases[2] which showed distinct tags and dislocated portions of adenoid, I believe that incomplete operation accounts for most of the failures; and that, in most cases, these could be avoided by careful removal of adenoid tissue from the nasopharynx under direct visual control. Little is known, of course, as to the cause of lymphoid hypertrophy. The possibility of a biochemical factor has been suggested, and iodine therapy and correction of dietary errors have been found helpful. Nevertheless a certain number of cases may persist in showing hypertrophy of nasopharyngeal lymphoid tissue. For these, and for the eradication of lymphoid tissue from the tube itself, the irradiation therapy, advocated by Crowe and his co-workers, would seem desirable. Certainly in the majority of cases the conventional procedure of adenoidectomy has produced satisfactory results.

Other causes of conduction or obstructive deafness for which intelligent treatment is usually effective in improving hearing are sinusitis, allergy, acute and chronic nasopharyngitis, and nasopharyngeal cysts or tumors.

The attempt to classify progressive deafness as conduction or nerve type is often of little practical value from the standpoint of therapeusis. Of far greater importance is the probable etiology. We must consider the possible causes such as obstruction, infection, toxic absorption, environment, acoustic trauma, nutritional and metabolic disorders, systemic disease, and the degenerative changes of advancing age. We must consider whether or not that condition is capable of reversal by our therapy, medical or surgical; and, most important, whether such reversal may improve the hearing or even arrest further impairment.

Local treatment by means of the eustachian catheter or bougie may be effective in certain cases of tubal obstruction, provided the underlying cause is not overlooked. Removal of infected tonsils or teeth, correction of nasal, sinal or nasopharyngeal pathology, or the eradication of more distant foci may result in improvement in hearing. These procedures should be applied only after careful observation and sound reasoning, based upon the history and the clinical and functional examinations. Proper management of cardio-vascular disorders, such as arteriosclerosis with hypertension, seemingly may improve hearing, perhaps by diminishing the tinnitus, possibly by improving the general well-being. The same holds true for many nutritional deficiency states and endocrine imbalances. But again any such treatment should be based upon definite indications brought out by careful study, and usually calls for the collaboration of consultants in other fields of medicine.

While certain writers have expressed enthusiasm for the specific treatment of hearing impairment by various drugs, often on an empirical basis, few, if any, impartial observers have been able to corroborate their results.

On the other hand our studies may indicate an already damaged cochlea, the result of acute or chronic infection or injury of the organ of Corti from drugs or acoustic trauma. Destroyed hair cells are not likely to be regenerated by any known therapy, conventional or otherwise. And, sad as it is, there seems to be no really effective therapy for the degenerative processes due to increasing old age, whether in the chochlea or elsewhere in the body. When hearing by bone conduction is badly impaired, any therapy other than amplification is at present only a forlorn hope.

We have long realized the futility of dealing with otosclerosis by our conventional methods. As yet we have accomplished nothing in the way of prevention, and our therapeutic efforts have been useless. A diagnosis, made only after careful study, advice as to future occupations and experiences, such as pregnancies, and a properly fitted hearing aid or lip reading are about all that conventional therapy has to offer.

Summary:

Our appraisal, then, would seem to indicate that conventional methods of treatment are effective when the conditions responsible for the hearing impairment are capable of reversal, and when therapy is applied before permanent and handicap-

ping damage has been done. For the most part obstructive deafness due to conditions in the nasopharynx, or in the external or middle ear, responds favorably. The eradication of foci of infection is more apt to be of benefit in the prevention rather than the improvement of already existing hearing impairment. Loss of acuity resulting from middle ear suppuration sometimes may be helped materially by prostheses. Local treatment of the eustachian tube, together with measures aimed at improvement in general health, may be beneficial but should be applied only on definite indications. Empiric or routine treatment, not based upon careful diagnosis, is rarely effective, and hardly can be classed as intelligent therapeusis.

In closing let me make this plea for the conventional procedures. Despite a natural dissatisfaction with what we have been able to accomplish, and a likewise natural desire for something better, indeed for as near perfection as possible; let us not be so swayed by enthusiasm for the new that we regard the old with a sort of glacial negation. In the field of prevention which, after all, is the big field, and in the care of the majority of our patients, the conventional will still have a place and rightly so, if based upon careful observation and logical reasoning.

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NON-SURGICAL TREATMENT FOR THE PREVENTION OF IMPAIRMENT OF HEARING

LeROY M. POLVOGT, M.D.
BY INVITATION
Baltimore, Md.

Members of the Otological Research Laboratory of the John Hopkins Hospital have been working for a number of years on the problems connected with the prevention of deafness. A high percentage of deafness results from infections in the middle ear and complications that arise from such infections. We believe that infected lymphoid tissue in the nasopharynx is the primary source of infections in the middle ear, nose and paranasal sinuses. It is with this belief in mind that in the prevention of deafness we focus our attack on the nasopharynx. As a result of our studies in 1940 we stated that the earliest impairment of hearing is the loss of high tones, and that this impairment may be caused by obstruction of the eustachian tubes by hypertrophied adenoid tissue.

In recent months there has been made available several therapeutic agents that may be used locally in the nasopharynx and the middle ear to prevent or clear up infections. These agents are: the chemotherapeutic agent, sulfadiazine; the bactericidal and bacteriostatic agents, tyrothricin and penicillin; and the radioactive agent, radon.

Pickrell was the first to develop a solution of sulfadiazine for local use. He used a solution of 2.5 per cent sulfadiazine in 8 per cent triethanolamine in the treatment of burns. This solution is absorbed by tissue and is non-toxic. The various toxic reactions associated with sulfonamides when taken by mouth are eliminated when it is used locally. This drug has been of special value in the treatment of ear infections when the infection was due to beta hemolytic streptococci. I stated in the beginning that most ear infections have their origin in infections of the nasopharynx. For two winters, studies have been made by members of the Otolaryngological Department of the Johns Hopkins Hospital on student nurses with acute coryza. Cultures were made from each student. The nose was sprayed thoroughly with sulfadiazine every hour the student was awake the first day; then every two hours the second to fifth day. Controlled studies indicate

that local treatment in this manner reduced the duration of coryza, lessened the disagreeable symptoms, and prevented complications such as otitis and sinusitis which though associated with acute coryza are not due to invasion of the tissues by a virus but rather by pyogenic organisms. Controlled studies also indicated that in acute suppurative otitis media, a 2.5 per cent solution of sulfadiazine used locally, shortens the duration of the infection. The discharging ear was cleaned and a medicine dropperful of sulfadiazine solution was put in the external auditory canal every two hours. The infection usually cleared up in about forty-eight hours. This result was not found in cases of chronic otitis media especially when the mastoid process was also involved. In such cases the infected mastoid process was cleaned out surgically and a generous amount of sulfathiazole powder put in the wound. Healing took place rapidly and with no evidence of infection of the wound. Any agent used in the middle ear and mastoid to combat and shorten the duration of an infection will lessen the possibility of the formation of adhesions, granulations, and changes in the drum, and will tend to prevent impairment of hearing due to these middle ear changes.

Rene Dubos in 1939 found that from Bacillus brevis, an aerobic sporulating soil bacterium, a specific bactericidal agent could be obtained. Peptone cultures of this organism were made and a water soluble and a water insoluble fraction obtained. The water insoluble fraction is extremely bactericidal. This material is called tyrothricin. Tyrothricin has been further fractioned into gramicidin and tyrocidine. Gramicidin is bactericidal for gram-positive organisms in general. It is believed to interfere with one or more metabolic systems of the individual bacterium. Tyrocidine works as a general protoplasmic poison and is bactericidal for both gram-positive and gram-negative organisms. It is usually made up in a 2 per cent alcoholic solution. One cc. of this solution in 100 cc. of sterile water gives a 1 to 5,000 suspension; a safe concentration for clinical use. The drug is toxic when used intravenously or subcutaneously but harmless when used as a contact agent. These agents in the proper concentration may be used directly in the middle ear and nasopharynx. In using these solutions the middle ear should be thoroughly cleaned and then filled with the solution two or three times a day. Packs soaked in the solution may be placed in the nasopharynx and allowed to remain for about thirty minutes twice a day.

In 1929 Fleming observed that the common bread mould, Penicillium, found on bacterial plates as a contaminant, inhibited bacterial growth. Penicillium is easily grown at room temperature on liquid amino acid media. Its greatest bactericidal activity occurs at a pH of 7 to 7.5 and it must be harvested when this pH is reached. The underlying liquid is poured through a seitz filter. The filtrate contains the crude bactericidal agent, penicillin. In liquid form penicillin is very unstable but when frozen its activity is maintained for about two weeks. The dry concentrate is more stable and is purified for intravenous injection.

Penicillin may be given by mouth, used locally or given intravenously. It is absorbed by the subcutaneous tissue, and the gut, and is excreted by the kidneys. Penicillin in general is bacteriostatic for gram-positive organisms and to a lesser extent for gram-negative bacteria.

The experimental work on the use of penicillin and tyrothricin in otolaryngology at the Johns Hopkins Hospital has been done chiefly by Crowe. He found that for a comprehensive and true estimation of the values of these drugs, it was necessary to make a complete bacteriological study of each individual case. This involved complete studies of each organism found on culture and its reactions to tyrothricin and to penicillin in various dilutions. A method of bacterial titration was used. It has been noted that the sensitivity of an organism to these drugs may change, that is the therapeutic agent may become unable to kill certain bacteria. It can be readily seen that to treat infections effectively with these agents, studies of bacterial sensitivity to them have to be constantly repeated.

Crowe believed the local use of these bactericidal agents in the middle ear would reduce the inflammatory reaction there and consequently the formation of granulation tissue. Therefore, the production of deafness by infection of the middle ear would be reduced.

One of the most useful agents in the prevention of deafness is radon. This agent has been in use sixteen years at the Johns Hopkins Hospital. It was first used

in a study of prevention of deafness in children. There is no type of impaired hearing that is peculiar to children. All types are found in both adults and children. Radon has proved very satisfactory in the treatment of impairment of hearing in all age groups. In order to hear normally it is imperative that the eustachian tube function normally. The most common cause for obstruction to the eustachian tubes is hypertrophy of lymphoid tissue around the orifices of the tubes. On account of its location it would be unwise to remove this tissue surgically. Radon will remove this tissue and clear the obstruction to the eustachian tubes. Recurrent otitis media due to chronic infections in the nasopharynx associated with hypertrophied lymphoid tissue can be prevented by treating the nasopharynx with radon and insufflating sulfathiazole powder into the middle ear. The use of radon for the removal of this tissue makes hospitalization, anesthesia, and surgical operation unnecessary.

SURGICAL TREATMENT OF IMPAIRED HEARING. THE RESULTS OBTAINED BY USING THE LEMPERT ENDAURAL FENESTRATION TECHNIC

MARVIN FISHER JONES, M.D.
New York

The most effective surgical procedure used to improve hearing to a serviceable level is the Lempert endaural fenestration operation. A definition of the word serviceable is important. Roughly, serviceable hearing is the ability to hear above 30 decibels in the conversational range. If the hearing is better than an average 30 decibel loss the fenestration operation does not offer a sufficient improvement to risk the possible dangers incurred in all operative procedures.

It was my belief, some six years ago, that the results of the fenestration operation, while dramatic, were not permanent. I know now that some results are permanent. I have seen patients whose hearing before operation, five years ago, was below the useful range and who can now hear low whispers in the operated ear. Unfortunately, this does not hold true for all patients. Some patients, who lose the improvement in hearing following operation, can again have their hearing restored by an additional operation or operations. A patient whose improvement in hearing has been maintained for one year without any diminution probably will maintain that improvement indefinitely. The hearing of some patients is not improved following operation and for some the hearing is made worse. When the statement is made, "The hearing was worse following the operation than it was before," one fact should be remembered; a patient who is accepted as suitable for operation has lost serviceable hearing. If the hearing is worse following operation, the difference may not be noticeable. Incidentally, the patients who are made worse constitute a small, but unknown percentage.

The endaural fenestration technic impressed me as being particularly dangerous. According to our orthodox teachings, it should be. However, actual experience has proved that serious complications are no more frequent following the fenestration operation than they are following other major surgical procedures used in otology. This seems an opportune moment to make some statements regarding the relation between complications and surgical ability. This is one operation that can not be bluffed. You can either do it or you can't! There is no middle ground. Fundamental knowledge and surgical ability are all important. An intimate knowledge of the anatomy and histology of the temporal bone is absolutely imperative. I can foresee one set of circumstances which will tend to discredit the fenestration operation. Those doctors who do not possess the natural surgical ability which is necessary to perform this operation, even though they may have had good training, will not get the best results. Only those doctors who have ability and are willing to spend months . . . yes, even years, perfecting the technic under competent instruction should attempt this most difficult surgical procedure.

I have wondered why the change was made from the old fenestration operation to the **Fenestra Nov-Ovalis.** My unwarranted deduction was, since the five year period of grace was up and the old cases had started to close, it was now time for a change in technic and a reprieve for another five years. I have examined patients on whom both operations were done. Some of the best results were obtained in the early days when the fistula was made in the horizontal canal. I have

seen the fistula itself macroscopically unchanged after five years.

In the discussion of the fenestra novovalis a little clarity regarding the use of terms will reconcile apparently divided opinions. The anatomical dome of the vestibule is visualized as the top of the vestibule when the patient is in the erect position. The surgical dome is the presenting part of the vestibule as the surgeon sees it when he is operating on a patient in the prone position.

Perhaps, enthusiasm has led to unfortunate claims regarding the amount of improvement in hearing and the percentage of patients who have obtained improvement. This enthusiasm is the normal reaction of any doctor who thinks he has added an important contribution to medicine. The reaction of the other doctors to over-enthusiasm should not obscure the fact that this operation does improve hearing from a point below useful levels to a point within a useful range. In a few instances the hearing has been restored to normal.

The whole subject of the fenestration operation has been distorted by conflicting uninformed opinions. Progress toward intelligent understanding has been obstructed by a fog of prejudice. Individual animosities, statements true and false, the emotionally unstable patient, the psychological factor always present in subjective recording of hearing, the pitiful vulnerability of the deafened to charlatanism, the reaction of despair when the expected results from a major operation are not forthcoming . . . all these factors have made reasonable and accurate conclusions by honest observers most difficult. Even the normal enthusiasm of any one of us when we have made a supposed discovery, leads us to wishful statements which time disproves. Here is a situation which viewed from the layman's viewpoint would be considered under the head of miracles, "making the blind to see and the deaf to hear"! There are bound to be many emotional misstatements of either an innocent or vicious nature. Time and study are still, and for some time, will continue to be the most important factors in determining which patients can be improved and which patients must wait for future developments.

I have seen patients who were unable to hear before operation, whose bone conduction was greatly diminished, and I have seen their records, made by competent members of our national societies. They were told in writing that they had nerve deafness. Some of these patients have insisted on operation and following the fenestration their hearing was restored to useful levels. These patients are, of course, exceptions to the rule. Similarily, I have also seen patients who had a diminished high tone perception (considered one of the cardinal signs of nerve deafness) whose hearing returned to normal after restoration of the patency of a closed eustachian tube. Our present methods of diagnosis would seem to be in error. Those of us who have studied histologic anatomy and pathology know how apt we are to make a diagnosis of clinical otosclerosis when the pathological examination shows none of the changes characteristic of this disease. The reverse is also true; i.e. pathological otosclerosis may be found without any symptoms having been present during life. Some years ago when visiting Dr. Crowe's laboratory at Johns Hopkins, I made the statement, "Our nomenclature and conception of the diseases causing deafness should be revised." The necessity of such a revision has been more firmly established through subsequent years of experience. If this operation does nothing more than shake our tempered convictions regarding the diagnoses and causes of deafness, it will have been more than worthwhile. We must go over all our problems, starting by changing our conception of fundamentals and develop the open minds essential to a solution of the impaired hearing problem.

The physiology of the vestibular apparatus, the chemistry of its most important fluids, the osmotic power of the endolymph membranes, the character of stimulations which activate the acoustic and labyrinth end-organs are but skeletal suggestions for a start. Much progress is being made. Many false conceptions have been discarded. Now a mechanical opening of the peri-lymphatic cavities accomplished by a method which at one time was considered hopelessly dangerous . . . has stimulated work along potentially productive lines. Again, I say if nothing more than this has been accomplished by the fenestration operation, it would still be a most notable advance.

Up to this point this paper has dealt largely with generalities, personal experience and personal observation. In order

to obtain impersonal factual data, I have sent letters to every otologist known to me as having done the fenestration operation. I have asked his period of training before attempting the operation, from whom he received his training, the number of cadaver operations he did before performing the fenestration operation on the human, any experiences he may have had doing fenestrations on animals and lastly, a record of his results. I have published this request in the Academy TRANSACTIONS, which as you know, is sent to a major portion of recognized otologists in the United States and Canada. The remainder of this paper will be based on the replies to these reports.

A few criticisms to clarify the atmosphere: The statement has been made, "The operation will not stop the progress of the disease known as otosclerosis." This statement is probably true, although there is no available proof one way or the other. The operation is simply a mechanical opening through the bony labyrinthine capsule with destruction of the endosteum. The peri-lymphatic fluid does not escape from the opening. When the fenestration operation has been completed a tissue which is flexible replaces a solid bony wall. A positive fistula test proves the foregoing statement and also shows how long the fenestrum stays open. I have seen the fenestrum nearly closed, leaving an opening so small that the fistula test was positive but that sound vibrations failed to penetrate. I have seen a lid of reformed bone removed from the opening and the hearing was again restored. This phenomenon indicates that, regardless of whether the bone disease of otosclerosis progresses or not, the hearing remains good following fenestration. The one known exception is when the disease actually invades the pathways of the nervous mechanism. Another criticism is, "The operation has been done on hopeless cases of nerve deafness." A few patients who have had a diagnosis made of nerve deafness have had the fenestration operation performed. The findings of the otologists who examined the patient before operation and made the diagnoses are a matter of record. These patients were told that the operation offered no hope for them. They persisted in their desire for operation. Following the operation their hearing was restored to a useful range. However, the fenestration operation should not be recommended for patients in whom the acoustic nerve is not functioning, unless the patient is fully informed that the return of hearing is not to be expected. Another criticism is: "No one has seen as many otosclerosis cases as the surgeons performing this operation have reported." In answer to this criticism, which is honest, I would say that the sooner we call a combination of symptoms known as otosclerosis, by some other name, the quicker we will eliminate a lot of unnecessary quibbling. No one knows whether patients will get temporary or permanent relief following operation. Some patients were considered most ideal subjects and they obtained no improvement in hearing. Other patients having symptoms considered by otologists as contraindicating operation have had a return to normal hearing. The name, otosclerosis, like Ménière's disease is not a specific identifying term.

A criticism has been made about promises to patients. In this, I am quite definite. No patient should be promised an improvement in hearing. No patient should be be told that the procedure is without danger. There is still danger in all surgical procedures. The danger of infection is minimized by flawless technic used preoperatively, during the operation and post-operatively in the fenestration procedure. Accidents have occurred and will continue to occur. They should be reported. The patient should be told about the vertigo which is an uncomfortable transient sequel to the operation. Sometimes the vertigo is quite persistent. Transient facial paralysis occurs with relative frequency. Only one case of permanent facial paralysis was reported to me. All patients should also be cautioned against swimming, using hot or cold water in the ears, subjecting themselves to cold draughts and excess violence in blowing their noses.

The fenestration operation should occupy a place in otology similar to other highly specialized surgical procedures. The development of the laryngectomy technic serves as a good precedent. The surgical perfection of today is the result of over twenty-five years intensive study and practice. At first very few laryngologists attempted laryngectomies, and even today the operation is done by a comparatively small number of nose and throat specialists. It seems reasonable to suppose that the development of the fenestration technic will follow a similar pattern.

Among the twenty-five doctors known to me as having been properly trained, there are only three who do not contemplate continuing the operation. These doctors stated that they believed the operation did produce good results, but for logical reasons they did not wish to do it themselves.

The number of operations performed by properly trained specialists is surprisingly small. Only six of the doctors reporting have done over twenty-five operations. The total number of patients who have had this operation performed is approximately fifteen hundred. Of this number approximately twelve hundred were done by two men. From these brief statements it will be evident that any percentage based on averages would be most misleading. More time and further reports will be necessary in order to furnish factual information or reliable statistics based on the experience of capable surgeons.

All the doctors reporting their experiences to me were refreshingly frank and the information they furnished was most helpful in the preparation of this paper.

Conclusions:

1. The **Lempert Endaural Fenestration Operation** improves hearing from a level below the useful range to a useful level.
2. I consider the five year period an adequate one on which to base the statement that some patients have had a permanent improvement in hearing.
3. If there has been no regression in hearing for one year following operation, the restoration of hearing will probably be permanent.
4. In general it may be stated that the results obtained by the fenestration operation will be in direct proportion to the ability, training, and experience of the surgeon.

The Academy and its Committee on the Conservation of Hearing has done well to arrange this program. It is time that a dispassionate and scientific survey of the subject, "What can be done for the patient with impaired hearing?" was made. I am sure a reasonable assignment of patients by doctors to groups suitable for operation, hearing devices, re-education and medical care can be accomplished.

USE OF HEARING AIDS

WALTER HUGHSON, M.D.
Abington, Pa.
and
SCOTT N. REGER, Ph.D.
Iowa City, Iowa

BY INVITATION

In any symposium on the therapy of deafness it is inevitable that the discussion on the use of hearing aids should come last upon the list of available procedures. But that the hearing aid should be considered as a therapeutic measure is a very considerable advance in our approach to the total problem of hearing loss and its alleviation.

Discussion of medical and surgical therapy is apt to lay emphasis on the method employed and its percentage of favorable results. If a certain medical routine, whatever it may be, exhibits some gain in hearing after a reasonable length of time it is considered successful. If following some surgical procedure the patient obtains a specified gain in hearing this too is successful. But in either instance the major concern is the decibel gain in improvement rather than the patient's actual psychologic and otologic reaction to therapy.

There can be no question that our individual efforts as otologists are tinged with a very definite personal bias. There is no particular reason why this phase of the problem should be labored further. Authoritative discussions have just been presented upon routine otologic treatment of hearing impairment, modern control of infections of the upper respiratory and auditory apparatus — unavailable to the practitioner — and finally the surgery of deafness. This last is really the most sensational thing that has been presented in the past decade.

However, by clinical diagnostic criteria, and standards of selection as to favorable cases we may assume conservatively that 80 per cent of the hard of hearing population of the country are not amenable to the help which any of these medical and surgical procedures may offer.

What then can the otologist suggest? The answer is an obvious and immediate one — namely the use of a hearing aid.

From the Otological Research Laboratory, Abington Memorial Hospital, Abington, Pa., and the Department of Otolaryngology, University of Iowa, Iowa City, Iowa.

Routine treatment of a hearing loss existant for one or more years in an individual in the third or fourth decade of life and exhibiting a loss of 40 decibels below normal threshold seldom results in any significant beneficial change in hearing. Under favorable conditions such a loss may react sensationally to modern surgical technics. But in neither instance can there be any actual promise of benefit.

Let us return, briefly, to the individual's reaction to his disability. The psychology of deafness is of profound and fundamental importance in our entire approach to this very significant problem. Improvement in hearing of an important degree is the difference between normal reaction and any degree between slight and profound depression.

Granted that every means at our disposal should be employed before we insist on the use of artificial aid for the improvement of hearing, when this point has been reached no alternative exists. A properly fitted hearing aid is the only answer to the problem. The benefit is immediate, the measure of gain can be a matter of record, and the cost involved is insignificant in relation to the specific performance of the instrument selected. Here we have a procedure which carries with it a guarantee of expected gain.

This discussion permits of nothing but a demonstration — an immediate one — of what may be expected from the use of a properly fitted hearing aid. Without such help the patient is a handicapped individual, with it he has hearing competence.

The otologist must assume responsibility for fitting hearing aids. Speech reception is the desideratum — by such technics as can be demonstrated to achieve this aim readily. We stress here as we have elsewhere that a hearing aid is fundamentally a form of treatment. As in materia medica, affinities and incompatibilities exist. This analogy may be slightly overdrawn but the successful fitting of a hearing aid depends to a large extent upon our knowledge of the expected reaction of a patient to one of these electrical devices. With all available refinements of technic in the fitting of hearing aids the utmost so far obtained is satisfaction in 80 per cent of all cases. This number may be increased to 90 per cent of success, a very high proportion for any form of treatment. It must be remembered that successful fitting here means the achievement of useful hearing, not simply a designated audiometric gain of a specified number of decibels.

Hearing aids are available which compensate for certain defects in hearing both in relation to the extent of loss and frequency emphasis. But there can be no set rule for this compensation. The patient requires an adequate gain in his ability to understand speech. Certain relationships have already been established between the discrete frequency audiogram and normal speech reception levels. The technic described is immediately applicable to the fitting of hearing aids.

Ideally, soundproofed rooms should be available and an adequately controlled electrical sound source. Such a system will be demonstrated, without of course, the benefit of a quiet environment. This equipment is not generally available and less technical procedures must be used if the fitting of hearing aids is to become an integral part of otologic practice. For this purpose an attenuated megaphone has been developed which for all practical purposes offers a reasonably accurate means of measuring the gain for speech which any hearing aid actually develops.

The entire procedure is time consuming and great patience on the part of the otologist is required, but the final results more than compensate for all effort expended.

Eighty per cent at least of hard of hearing individuals acquire from this none too rigorous and relatively brief ordeal — five hours at the most — a gain in hearing which places them at a relatively normal hearing level.

There is of course a training period. The otologist's interest and support makes this period a comparatively simple one. No objection can be raised to continued medical or surgical treatment after the adoption of a hearing aid. It can be laid aside if happily therapy should make its use unnecessary. But the patient has learned that he has this ready bulwark against possible periods of auditory failure. If treatment is unavailing the solution of the entire problem is at hand.

In Memoriam

LEE WALLACE DEAN, M.D.
1873-1944

LEE WALLACE DEAN, M.D.

1873-1944

Lee Wallace Dean, professor emeritus of otolaryngology, Washington University School of Medicine, St. Louis, and a former President of the American Academy of Ophthalmology and Otolaryngology, passed away February 9, 1944.

Born in Muscatine, Iowa, March 28, 1873, Dr. Dean studied at the University of Iowa and received his degree of doctor of medicine there in 1896. After studying in Vienna a year he returned to Iowa to practice medicine. At the State University of Iowa, Dr. Dean served as professor and head of the department of otolaryngology and oral surgeon from 1900 to 1927. From 1912 to 1927 he also served as dean of the medical school. He then went to St. Louis as professor of otolaryngology of Washington University School of Medicine and subsequently he became director in research in otolaryngology at the Oscar Johnson Research Institute.

Besides being otolaryngologist-in-chief at the McMillan Eye, Ear and Throat Hospital, he was a member of the staff of that institution and also of Barnes Hospital, St. Louis Children's Hospital, Jewish Hospital and St. Louis Maternity Hospital. He was otolaryngologist-in-chief to outpatients at the University Clinics.

Dr. Dean was a Life Fellow in the American Academy of Ophthalmology and Otolaryngology, having been elected to membership in 1898. He served as President in 1937. He was also at one time president of the Iowa State Medical Society, American Laryngological Association, American Laryngological, Rhinological and Otological Society, American Otological Society and secretary of the Section on Laryngology, Otology and Rhinology of the American Medical Association. Dr. Dean was a member of other scientific groups including the Association of American Peroral Endoscopists, Missouri State Medical Association, American Broncho-Esophagological Association, and La Société de laryngologie des hôpitaux de Paris.

During World War I, he was commanding officer of General Hospital No. 54 and was a lieutenant colonel in the medical officers reserve corps, retired. In 1937, he was honored with the de Roaldes Gold Medal of the American Laryngological Association and from 1927 until the time of his death, Dr. Dean was editor-in-chief of the Annals of Otology, Rhinology and Laryngology.

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Editorial

The Academy

The necessity for continuation courses of study in the medical specialties has never been more urgent than now. Not only are physicians in civil practice compelled to enlarge their field of activity and to refresh their knowledge of the intricacies of modern diagnosis and treatment, but the doctors in military service are also meeting with problems of medical management that they must solve themselves. Men of both groups, some whose preparation for the best handling of the patients they must care for has not been completed to their satisfaction, are eagerly subscribing to every available opportunity to add to their skill and knowledge by reading books and journals, by visiting clinics, and by attending meetings of scientific societies. The demand for all means of instruction is greater than it has ever been before, and the present efforts of teachers in both civil and military personnel has never been exceeded.

For nearly fifty years the Academy has successfully sponsored medical education by publication of scientific papers on timely subjects in ophthalmology and otolaryngology. For more than twenty years it has provided courses of lectures and demonstrations in the Section on Instruction, and for five years has carried on Home Study Courses that have proven their value by being oversubscribed every year since they were started. The value of these Home Study Courses is recognized by the Specialty Boards and they are recommended for residents and candidates for board certification, although it is specifically understood that completion of the Academy courses does not necessarily fulfill the requirements of the Boards for fundamental training.

These educational projects of the Academy are established on the soundest principles of modern teaching and have pioneered the frontier of practical medical requirements without a single failure. They have been adopted in principle and in practice by organizations in other medical specialties and their value has been enhanced by almost universal adoption in clinical societies.

The members of the Academy and invited lecturers who give so generously of their time to participate in the instructional programs constitute a large and distinguished faculty in medicine. It is

an outstanding attribute of the American way of life—service for humanity without thought of recompense or material reward.

Annual Convention

The forty-ninth annual meeting of the Academy will be held in Chicago, October 8-12, 1944, at the Palmer House. Everyone is familiar with present day conditions as regards travel and hotel facilities but here is a reminder: Make your hotel reservations NOW and secure your railroad and Pullman TICKETS thirty days before the opening of the meeting.

The essentiality of the annual meeting of the Academy is unquestioned by any governmental agency. You are to be your own judge as to what it means to you and to govern your action accordingly.

The scientific program has been extended to permit more lectures, demonstrations, instructional periods, motion pictures, committee meetings, technical and scientific exhibits than ever before in the history of the Academy. The country has been combed for specialists in the latest development in diagnosis and treatment of diseases of the head and the organs of special sense as well as for projects in research in visual and auditory function, in chemotherapy and modern surgical technic. Special programs are being prepared by the Committee on Industrial Ophthalmology and by the Commitee on Conservation of Hearing. The activities of these Academy committees cover an enormous territory and affect every doctor of medicine.

The complete program will be published in the August issue of the TRANSACTIONS.

The Directory

The Directory was published last year as a supplement to the TRANSACTIONS for the convenience of the Fellows. In this form it permits the use of a desk copy without disturbance of the regular file. The innovation met with universal approval so will be repeated this year. Approximately 3,500 names and addresses, corrected to April 1, 1944, are included. Due to many changes necessitated by change of address, zone designation, military service and fellowship classification it became necessary to select new format and type. The home office will appreciate your comments.

Letter to the Editor

February 8, 1944

Dear Doctor Benedict:

The literature on biomicroscopy often mentions the name "slit lamp," which, however, does not correctly describe the instrument as it is used today by the ophthalmologist.

Some people call it "biomicroscope," but that is not the correct name either, since an ordinary binocular microscope used in biology in examining specimens like protozoa, etc., is also a biomicroscope, but, of course, the illumination is the ordinary illumination as used with any microscope.

I, therefore, contemplate to change the name in our literature to "Biomicroscope with Slit Lamp Illumination" as a distinction from the ordinary microscope used in biology for examining of living specimens.

No doubt you know that the first slit lamp developed by Professor Gullstrand and made by Carl Zeiss, Jena, was a single slit lamp mounted on an individual base, not combined with a microscope. This simple slit lamp was used on

a table in conjunction with the well known corneal microscope as two separate instruments. Later on the two instruments were combined, but the name "slit lamp" still remained and is still often used. I, therefore, should like to have your opinion whether my suggestion, as expressed above, meets with your approval.

Trusting that I do not trespass too much on your kindness with this question, I remain,

Very cordially yours,

MAX POSER

Alumni Dinners

In setting up arrangements with the Palmer House for the annual convention the hundreds of items that are concerned with the smooth running of the meeting must be anticipated and catalogued. No sooner is one convention concluded than plans for the next meeting begin to take definite form. The Program Committee met in January and laid the groundwork for the meeting that will be held in October. It was evident at the last meeting that more time is required to stage all of the Academy activities so the meeting this fall will cover one more day — five in all.

Changing restrictions in the food and beverage departments of the hotel make it impossible to select menus until just before the week of the meeting although prices are agreed upon with a minimum of elasticity. Certain consumable products heretofore available on order are no longer obtainable and others are to be had only in limited amounts and under special conditions of service.

The alumni dinners have been arranged by the Board of Secretaries for the convenience of the Fellows. The cost of tickets, the tips, the tax and in some cases loss because of misunderstanding has been charged to convention expense. Misunderstanding is the only reduceable item. The Secretary's office must be informed of the wishes of the various alumni groups if the customary dinners are to be continued. Each group must plan its own program of activities and inform the Secretary of the Academy of its needs in way of space and the number expected to attend. Please do this at once without personal solicitation from the home office. *Fees collected from alumni will be for restaurant service only.*

Plaqued Certificates

Due to the inability to obtain proper materials, further orders for plaqued certificates will not be taken. Orders that have been accepted will be filled in order of their receipt as rapidly as they can be procured.

AMERICAN ASSOCIATION OF EYE, EAR, NOSE AND THROAT SOCIETY SECRETARIES

W. Howard Morrison, M.D., *President*
Kenneth L. Craft, M.D., *Secretary-Treasurer*

The annual business meeting of the officers and directors of the American Association of Eye, Ear, Nose and Throat Society Secretaries was held at breakfast on Monday morning, October 11, 1943, at the Palmer House, Chicago. Among those present were Drs. D. S. DeStio, Pittsburgh; W. H. Morrison, Omaha; L. A. Brown, Atlanta; K. L. Craft, Indianapolis; and Sam H. Sanders, Memphis.

Among the matters discussed at length was that of attempting to secure a more favorable time for holding the annual meeting of the Association. Many complaints have been received from the members to the effect that the present plan of holding the meetings at a noon luncheon allows too little time for the proper conduct of the meeting and for discussion of the program and business features. Much enthusiasm has been displayed by the membership over the general purposes and ideals of this organization but interest has waned and further development has been curbed because of lack of time at the meetings for fuller expression and interchange of

The officers and directors are working upon this problem and it is hoped that thoughts and ideas. This has been the one outstanding criticism heard from all sides.

Editor's Note. — The Program Committee of the American Academy of Ophthalmology and Otolaryngology has arranged for the American Association of Eye, Ear, Nose and Throat Society Secretaries to hold a dinner meeting at 5 o'clock, Wednesday, October 11, 1944. This will allow ample time for business matters and discussion, and it will not conflict with any features of the Academy program.

W. L. Benedict, *Editor-in-Chief*

a satisfactory solution will have been reached by the time the next meeting is held. Meanwhile, comments and suggestions upon this matter are welcome.

The American Association of Eye, Ear, Nose and Throat Society Secretaries is now of sufficient age, size and background to merit a constitution and set of by-laws to govern its actions and administrations. A committee appointed for this purpose is working out the details and will present the proposed constitution and by-laws for consideration at the next general meeting. This is an important step. The officers and directors have been forced of necessity to make decisions in a most arbitrary and undemocratic manner during the organization and development of this society. They realize that this has been irregular and not quite fair to the rest of the membership and will welcome the adoption of a specific code of rules upon which the government of the society affairs can be based.

Officers and directors for the coming season were chosen as follows:

Officers

President—Dr. W. H. Morrison, Omaha, Neb.
Pres.-Elect—Dr. Paul Craig, Reading, Pa.
Sec.-Treas.—Dr. Kenneth L. Craft, Indianapolis, Ind.
Advisory Council—Dr. W. H. Theobald, Chicago, Ill.

Directors

District No. 1—Dr. Burton Lovesey, Boston, Mass.
District No. 2—Dr. S. B. Freeman, Buffalo, N. Y.
District No. 3—Dr. L. A. Brown, Atlanta, Ga.

District No. 4—Dr. Sam H. Sanders, Memphis, Tenn.
District No. 5—Dr. D. S. DeStio, Pittsburgh, Pa.
District No. 6—Dr. Vernon Leech, Chicago, Ill.
District No. 7—Dr. W. E. Keith, Kansas City, Mo.
District No. 8—Dr. J. C. Ogden, Long Beach, Cal.

KENNETH L. CRAFT, M.D., *Secretary*
1002 Hume-Mansur Bldg.
Indianapolis 4, Ind.

Hints to Secretaries

PAUL C. CRAIG, M.D.
READING, PENNSYLVANIA

HOW CAN a secretary be of most value to his Society?

1. He should take care of correspondence promptly and efficiently.
2. He should send each speaker suggestions concerning the customs of his Society and make the necessary reservations for him.
3. He should send each speaker a thank-you note including a resume of the reactions of his members.
4. He should keep the Papers, Archives, and Minutes in good order.
5. He should keep a scrapbook of publicity pertaining to his Society.
6. He should maintain an extensive mailing list of persons potentially or actually interested in his organization.
7. He should send out mimeographed or printed invitations as early as possible, ten days and preferably two weeks before each meeting.
8. He should make it his business to contact as many speakers as possible and line them up tentatively. This will make the work of the program committee more satisfactory.
9. He should collect ideas from everywhere and put them at the disposal of the proper committee chairmen, but he should not push his own pets and make himself disagreeable.
10. He should help the program committee with the experience gained from his contacts with the American Association of Eye, Ear, Nose and Throat Society Secretaries. Thereby programs will be varied and ruts will not be established.
11. He should keep everybody interested by helping to plan rotating committee appointments. Committee members should be moved up in succeeding years depending upon their aptitude and versatility.
12. He should carry out the policies of his Board of Directors and not impose his own. Meanwhile he should use his knowledge of what is good and what is bad for his Society, assisting unobtrusively and imperceptibly.
13. He should see to it that disagreements will not reach general business meetings, and act as an impartial and conciliatory influence.
14. He should consult with the open-minded members of his Society and engage their help in cases of personality conflicts. This will tend to create larger vistas and enable him to carry out the broad policies of his Society.
15. He should give consideration to all suggestions from his members and not dismiss them just because they were unworkable elsewhere. The more suggestions the more results.
16. He should not be the "power behind the throne" and he should prevent any individual or group from becoming "the throne."
17. He should do the work and let others do the talking.
18. He should lean heavily on the American Association of Eye, Ear, Nose and Throat Society Secretaries, and he should consult frequently with the Secretary of that organization.

In Memoriam

George Lindsay Johnson ..... Natal, South Africa

Israel Grushlaw ..... New York, New York
December 9, 1943

John W. Millette ..... Dayton, Ohio
December 28, 1943

Ernest Reed Hirst ..... Camden, New Jersey
January, 1944

Will Otto Bell ..... Seattle, Washington
February, 1944

Lee Wallace Dean ..... St. Louis, Missouri
February 9, 1944

William W. Pearson ..... Des Moines, Iowa
February 11, 1944

Sanford R. Gifford ..... Chicago, Illinois
February 25, 1944

Adolph O. Pfingst ..... Louisville, Kentucky
February 25, 1944

AN APPRAISAL OF THE VALUE OF AN ORTHOPTIC CLINIC IN PRIVATE PRACTICE

E. C. Ellett, M.D., and R. O. Rychener, M.D.
Jean S. Robinson, A.B.
BY INVITATION
MEMPHIS, TENNESSEE

The Memphis Orthoptic Clinic, available to all ophthalmologists in Memphis, has been in operation for ten years. It was organized in a frank spirit of skepticism, and because of this feeling, its facilities have been available to our patients without special reference to expense, thereby allowing us to refer to it any type of muscular condition for treatment as long as desirable. The records of the clinic have been studied to determine whether it has justified its existence.

In the last five years, 958 patients have been seen at least once for examination and diagnosis. A large number were patients with whom we were able to maintain personal contact. Of this latter group, 345 were found suitable for orthoptic training and have come at varying periods for such training. In the past year, 389 patients were seen in the clinic, of whom 334 were suitable for training, indicating a very healthy increase in our appreciation of what an orthoptic clinic can do for private patients. At the present time 53 patients are receiving regular office treatment for half-hour periods, three times weekly.

The clinic is operated by two trained technicians, of whom one (J.S.R.) is certified by the American Board of Orthoptic Technicians. Were space and talent available, the number of attendants could well be increased.

Presented at the Forty-Eighth Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 10-13, 1943, Chicago, Illinois.

The first step in orthoptic work is the investigation of the state of muscle balance—for which the screen test made with prisms, for near and far distance and in the cardinal fields is of prime importance. For the testing of fusion ability and projection the instrument most necesary is one of the major amblyoscopes. Other useful instruments are the Synoptophore, the Synoptiscope, and the Orthoptoscope. Of these the Synoptophore is the one with which we are most familiar. Its adaptability and the clear view of the patient's eyes that it affords make an instrument of this type invaluable in treatment as well as necessary in diagnosis. In its favor is the fact that it is not automatic, thus facilitating fine adjustments. It is by means of the major amblyoscope that abnormal retinal correspondence is most easily detected, and it is with this instrument only that cases of this type are treated binocularly.

A simple hand stereoscope should always be available for the development of fusion amplitude and stereopsis. It is not useful in many manifest conditions, and under no circumstances should it be used when abnormal retinal correspondence is suspected. However, generally speaking, in a high percentage of cases that require orthoptic treatment, a stereoscope will be of value for both office and home treatment.

The Junior Metronoscope, equipped with rotary prisms, is an instrument which we have found to be useful in many ways. We would recommend it particularly for treatment at reading

distance. Persons with convergence insufficiency are very quickly benefited by prism reading. When suppression is suspected or known to be slight, the Metronoscope is made more useful by the addition of a reading bar. It serves a very real diagnostic purpose in tests for convergence insufficiency.

In our clinic we also make use of the following apparatus:

1. The standard phorometer with Risley prisms, plus a Project-O-Chart for determining muscle balance.
2. The Stereo-Orthopter, an automatic instrument with lateral motion, excellent for developing fusion amplitude and for training stereopsis.
3. The Maddox Cheiroscope, used for suppression cases and for those whose binocular coordination is true, but weak.
4. The Kinetic Stereoscope, designed for rotary movement, guided by hand.
5. The Remy Separator, a simple easily made hand device useful in many cases of lateral imbalance.
6. A reading bar of the simplest design which is of distinct use for many cases of binocular imbalance where voluntary control is being taught.
7. We find many uses for a red and blue filter; in recession exercises with a light, and in suppresion cases in connection with red drawings to trace.

When one begins the examination of a patient with a variety of symptoms, the routine tests should at least be of such an extent that any gross error in muscle balance is soon detected. Tests ordinarily made are for visual acuity for far and near, a quick screen and parallax test, measurement of the near point of convergence, and an estimate of the muscle balance for 20′ and 14″, with vertical prisms or the Maddox rod. Usually correction of errors of refraction, when indicated by faulty vision or symptoms, gives the desired relief. In cases of squint or any more than what may be called a normal amount of heterophoria, further tests of binocular vision, fusion and muscle strength (prism divergence and convergence) are made, and if the conditions seem to justify it, or if symptoms of asthenopia persist after a careful correction of the refraction and wearing of the glasses, the patient is referred for further study in the orthoptic clinic. It would be easy to tax the resources of the clinic by sending more people there, but the plan we follow gives us a chance to estimate the need of such tests and saves the time of the clinic workers. In other words, we do not try to call on the clinic for such information as we should properly and easily acquire with the equipment in the trial case, nor do we hope to have the clinic look after patients who need only a proper refraction or some form of local or general medical treatment.

The role of orthoptics in the treatment of binocular incoordination is unique in that there is no substitute for the particular part that it plays. Just as refraction has its own place, so does orthoptic treatment. The realization that the miraculous cannot be accomplished by this comparatively simple procedure will lessen the disappointment of those whose expectations are too great.

We do not make extravagant claims of progress made through orthoptic treatment, but by rather cautious consideration we are gradually coming to more definite conclusions about the value of such treatment. Two quotations from recent articles agree with our own experience and conclusions. Linksz states:

"Orthoptics was rendered a poor service by those early enthusiasts who defined it as the nonoperative, as opposed to the operative treatment of squint. There is but one way of treating squint; namely, by the purposeful integration of a number of different pro-

cedures of which surgery, while the most dramatic, is but one, and orthoptics another."

Lancaster reports:

"Orthoptics is not exercising of the ocular muscles; it is not even primarily a procedure designed to straighten the eyes. It is the teaching of a patient to use his two eyes together for comfortable binocular vision. The emphasis in such training is the teaching of a skill in the use of unskilled neuromuscular coordination. Orthoptics will be of much greater value to physicians and their patients when its function is re-evaluated; not as primarily concerned with anomalies of eye position, but as devoted to helping the patient learn comfortable binocular visual habits."

Our first efforts in orthoptic therapeutics were directed entirely toward the correction of squint in young individuals. We soon discovered that we could not obtain improvement in coordination by exercises alone, but that correction of errors of refraction by glasses and gross deviations by surgery were necessary before much effect from orthoptic training could be realized. In addition, amblyopia must first be combatted by occlusion, and in our experience only complete occlusion of the fixing eye over a period of months has ever given any results. An exception to complete occlusion is made in case of accommodative squint, where the stippling or lacquering of one lens is often desirable in an effort to overcome suppression. We have many records which show that visual acuity in an amblyopic eye has increased from 6/60 to 6/6 and remained so following removal of the occluder. On the other hand, we have had many patients whose vision could never be improved beyond 6/15, and in whom there was no reason for failure to improve apparent by ophthalmoscopic examination. Unless optimum visual acuity is 6/12 or better, our experience indicates that the ultimate goal of normal position of the eyes and third degree fusion cannot be reached.

Among our records are two outstanding cases where glasses were not indicated and orthoptic training accomplished normal coordination of the eyes. The first was a case of convergence excess. H. W. was a patient of Dr. J. B. Stanford, who has kindly given us permission to report his case in detail.

The child, H. W., was four years of age and wore no glasses, the refractive error being nil under homatropine cycloplegia. The visual acuity was 6/7.5 in each eye. He could hold his eyes parallel for distance, but on

fixation of any object within a range of three feet, his left eye converged about 35△. Fortunately, he was aware of diplopia as soon as his eye turned in and he would immediately make an effort to straighten it, but it was an

impossible accomplishment for near. He was placed on daily orthoptic training, and at the end of several months was able to read and draw, with the eyes held straight. He has continued to use the stereoscope at home and will undoubtedly require continued supervised training at intervals for several more years.

The second patient, M. T., aged four years, was one upon whom one of us (R.O.R.) had successfully operated for alternating convergent squint of 50△. Glasses were never worn because the total refractive error under atropine cycloplegia was only +.50 sphere. Visual acuity was 6/6 in each eye. The eyes remained in perfect position for eight months, when, after suffering a fright during a tornado, there was an immediate convergence, with development of convergent squint of nearly the original amount. She was then placed under daily orthoptic training. She gradually improved, but would have alternately good and bad days. On bad days she measured a deviation of 38△ esophoria, 2△ left hyperphoria. On good days her eyes were perfectly straight. With regular treatment for a month she was able to hold her eyes parallel for several days at a time and was sent home with a stereoscope and simple fusion cards. She has been checked at intervals, and for the past year has shown no tendency whatsoever toward deviation from the normal.

ANOMALOUS RETINAL CORRESPONDENCE. Anomalous retinal correspondence, or false projection, is a difficult problem in the rehabilitation of patients with squint, and is best handled by the trained orthoptic technician.

In the past five years, 36 per cent of the squint patients examined in the orthoptic clinic had false projection. This agrees very well with the reports of others, i.e., Dr. W. T. Davis, 36 per cent; Miss M. A. Pugh, 50 per cent; Dr. T. B. Travers, 50-60 per cent; and Dr. LeGrand H. Hardy, 60 per cent.

False projection has proved to be one of the greatest stumbling blocks in the training of true binocular coordination. The importance of detecting this condition when the original diagnosis is made cannot be emphasized too strongly. Neglect in taking note of anomalous correspondence amounts to emphasizing the squint instead of alleviating the condition. The following cases will serve as illustrations of this statement:

(1) A. P. Alternating exophoria — divergence excess. Fusion was reported at 10△ exophoria, but at this angle on the Synoptophore or when corrected by prisms, there was definite shifting of fixation. She appeared to use an eccentric area of her right eye only when using her eyes binocularly, fixation of each eye separately being true. Shifting was eliminated by prisms at 22△ base in, and true fusion and stereopsis was finally seen at this angle. Had false projection been ignored, exercises would have been given at the false angle, thus intensifying the squint. Instead, however, all exercise at first was given at what appeared to be the true angle until fusion was obtained. After fusion was well fixed at the true angle, adduction exercises

were carried out, great care being given to observe that she did not revert to using her old false area. When treatment was stopped, she had true fusion and stereopsis and held her eyes straight. Since the condition was divergence excess, much depended on the control she exerted when looking at distance.

(2) L. M. The patient reported fusion at 0 and at 10Δ base out. The true angle appeared to be at 23Δ base out. The left eye projected falsely. Treatment was given at the true angle, and the lower half of the left lens was also occluded to prevent binocular vision for near, but to encourage it for distance. It was not possible to say if the patch was used correctly, but the results were fairly good. Final result was true fusion at 8Δ esophoria — she could overcome this and use the eyes held straight.

(3) F. L. Fusion was reported at 10Δ base out, but the child was definitely exophoric. The true angle seemed to be 10Δ exophoria. Good fusion and stereopsis were developed at the true angle. The eyes were held straight and good convergence ability was developed. If the fact that the subjective angle was false had not been taken into consideration, exophoria would have been emphasized rather than overcome.

Particularly with operative cases we find that an abnormal correspondence which has existed before operation is a definite handicap in postoperative treatment. The patient has a tendency to want to revert to his old habit, and much valuable time is lost at this strategic moment — immediately after operation. Theoretically and ideally, it would seem the most advisable procedure would be occlusion to develop macular fixation of the squinting eye, then operation, then treatment. This, of course, for various reasons, is not possible in all cases.

We have the record of a patient (P. B.) whose case demonstrates what a handicap abnormal correspondence can be in postoperative training. He was an out-of-town patient and had been given no preoperative treatment. Immediately after operation, he reported fusion on the Synoptophore at 0 or 3Δ base in. At this angle shifting was seen, and it appeared that his true angle was nearer 10Δ base out. He returned to his home within a few days and there was no opportunity to give him any kind of binocular training. Had this been possible, however, treatment would have had to be given at his objective angle in an attempt to develop true fusion. At this particular time, had there not been the question of a falsely used area to consider, abduction exercises would have been indicated. The outcome in a case of this kind is often a cosmetic, rather than a functional, result.

Divergence Excess. It is not possible to make a general statement concerning patients with divergence excess, since so much depends on the individual. The tendency to diverge to excess at a distance usually remains, but the development of the ability to overcome this tendency is the purpose of orthoptic training. The factors of cooperation and mental effort on the part of the patient affect to a great extent the final result. Many of these patients require operation as well as orthoptic training.

The factor of mental effort is one that cannot be ignored in the successful treatment of any kind of binocular imbalance. Without the patient's full cooperation, and that cooperation includes a definite positive striving toward the goal explained by the doctor and kept in view by the technician, the achievement of the desired result will be decidedly hindered. This is true with patients of all ages.

The adult, who as a rule seeks relief from discomfort of some kind, should give his cooperation without question. Yet surprisingly many times he has to be persuaded to do so. We believe the best method with this adult-age group is the full explanation of the common sense of the treatment. The sound logic of orthoptic training coupled with its comparative ease and simplicity should appeal to the average intelligent adult.

In the case of the 'teen age patient the cosmetic result is usually the point of attraction. With the very small child the idea of showing what he can do seems to be the theme to stress. Some entertainment is necessary, and the child with imagination is the ideal. The method of reward, however wrong psychologically, must at times be the resort. Much ground is gained when the person working with the child can secure his affection and esteem. It should be stressed that securing this cooperation, by whatever means, is all important with patients of all ages.

In our retinal separation cases, we have with impunity severed extraocular muscles in order to expose the proper scleral area and in but few instances has there been any postoperative heterophoria. However, the report of a single patient will illustrate the aid which the orthoptic clinic can give in the treatment of such complications.

A. D. M., aged 65, suffered an enormous tear and detachment of the retina in December, 1937, for which diathermy operation was performed by one of us (R.O.R.) on January 7, 1938. The left superior and external recti muscles were reflected and then resutured to the stumps with fine chromic catgut. Following recovery of normal vision there was a pronounced divergence and hypertropia of the left eye. On the Synoptophore there was simultaneous macular perception, fusion and stereopsis at 24△ base in, with a left hypertropia of 7△ to 18△. Diplopia and vertigo were constant symptoms. After thirteen orthoptic treatments there was a marked improvement in ocular measurements to 10△ base in with a hyperphoria of 6△. He later overcame this defect by constant effort and obtained fusion and stereopsis at 0. He has maintained this ability to the present time with entire relief from his symptoms.

Divergence Insufficiency. There are many patients with divergence insufficiency who are made more comfortable when the power of prism divergence is built up. In the clinic are records of many who failed the preliminary army aviation tests because of weakness in diverging power, who were enabled through proper exercises to qualify for the Air Force. Included also are records of laboratory technicians who experienced difficulty in the use of the binocular microscope, and patients with headaches and other asthenopic symptoms for which no other cause than mild heterophoria could be discovered and who became perfectly comfortable after orthoptic training.

Convergence Insufficiency. In our experience those cases with the best prognosis and most rapid response to treatment are ones with convergence insufficiency. The treatment for the alleviation of this condition is admittedly simple, but it does not follow that this deficiency is any less worthy of notice. Patients suffering from a lack of a good convergence ability feel very real discomfort which, though not serious, can hamper them considerably in their work. Sometimes the symptoms are only with close work, whereas again discomfort is felt rather generally with use of the eyes at any distance. Office training of a better and easier convergence habit, supplemented by home exercise is the usual procedure. Many out-of-town patients suffering from this condition are seen. Happily for them very successful home treatment can be directed. Of course, as is the case in any kind of training process, much depends on the amount of effort put forth by the patient. It is true, however, that more can be done for himself by a patient needing convergence exercise than can be done for any other condition. It is probable that there are those who disparage treatment for convergence insufficiency, dismissing the sufferer with a half-hearted suggestion concerning finger to nose convergence exercises. There are also persons with a convergence weakness who are advised simply not to use their eyes for

much close work, and who inevitably become dissatisfied patients. We feel it urgent to bring these possibilities to mind since in our experience so many uncomfortable patients, although properly refracted, have been completely relieved by orthoptic training.

TRUE AND LATENT CONVERGENCE INSUFFICIENCY. In this field, we would like to differentiate between a true convergence insufficiency in which the diagnosis is apparent because of a marked recession of the convergence near point, a high exophoria for near or a frank disparagement in the normal 3 or 4 to 1 relationship of prism convergence to prism divergence as measured on the phorometer, and a latent or hidden tendency to convergence insufficiency as demonstrated by normal phorometer measurements accompanied by a history of ocular discomfort on close use of the eyes. The latter, we believe to be a large group of patients making the rounds of various ophthalmologists' offices, and usually thought to be the victims of neurasthenia or hysteria. Careful checking with the phorometer may disclose a slight weakness in prism convergence, but usually the muscle measurements will be found to be within normal limits. However, examination on the Junior Metronoscope equipped with rotary prisms will invariably disclose a poor reading ratio, i.e., the relationship of prism convergence to prism divergence which should be at least 2 to 1 and preferably 5 to 1 or 4 to 1. On this instrument are simulated the fixations and regressions, which are a part of the normal reading habit. Measurements of the vergences under the stress of movement of the eyes from left to right as in reading gives us a true measurement of muscle power in ocular activity as compared with the static measurement of unmoving eyes examined on the phorometer by fixation of a single point object. Since acquainting ourselves with the value of prism reading tests in such patients, we have looked more closely for the latent insufficiencies and naturally find them to occur more frequently in our practice than we would have believed possible.

The diagnosis of convergence insufficiency (greater exophoria for near than for distance, together with poor convergence), is often very easy to make. However, there is no definite rule that can be set up concerning exact measurements that lead to a diagnosis of this difficulty. First of all, symptoms vary greatly in intensity — consisting of headaches, double vision, tired eyes, burning lids, dizziness and actual unsteadiness when walking. We have found many combinations of symptoms as well as many different groupings of indicative measurements. There sometimes exists a decided exophoria for near with a good convergence while at other times there may be poor convergence with normal physiological exophoria. Either of these combinations may be present with good or poor prism vergences.

There is, however, a significant finding that in our experience consistently indicates the need for convergence training at the near point. This measurement is called the reading ratio. It is the ratio of prism divergence to prism convergence at the near point during actual reading, which involves the changing of fixation as is done normally during reading or during use of the eyes in almost any other occupation. In some few instances this measurement has been the only indication of the difficulty present. In other cases, it has been a deciding factor in the course of treatment. In all cases, it has borne out the testimony of other positive diagnostic measurements.

There may be several ways of setting up means of taking a reading ratio, but the method we have found most satis-

factory is with the use of a table model Junior Metronoscope. This is an instrument equipped with reading rolls lighted from within and with an automatic shutter arrangement whereby fixation is necessarily changed in order to read the complete line. There is also firmly attached a set of rotary prisms at reading distance complete with head rest. The patient places his head firmly against the rest wth his eyes close to the prisms. The reading correction is worn. As the prisms are turned base in and base out to find the breaking point, the patient changes his fixation as he reads across the line, guided by the opening and closing of the shutters. The resulting ratio is prism divergence over convergence—a measurement made more significant in this particular difficulty, since the patient is actually doing the thing with which he has trouble while the measurement is being made. We find the same relationship is necessary for comfort as is necessary between prism divergence and convergence at distance.

It can easily be seen that by means of this same instrument, the Metronoscope, the training of better convergence, as well as the diagnosis of convergence deficiency, can be very effectively given. We depend to a great extent on this instrument for the treatment of those suffering from this weakness. Other methods include the use of the Synoptophore, the Stereo-Orthopter, the Remy Separator and other devices.

In the past five year period, the clinic has recorded 453 cases of convergence insufficiency of whom 148 or 33 per cent are classified in the latent group.

Of 136 convergence cases of insufficiency treated in the clinic, 52, or 38 per cent, are classified as latent convergence insufficiency cases. Of the 136 treated, 125 were definitely improved. Of the 11 not improved, 5 did not complete the treatment. The remaining 6 had other conditions such as allergy, migraine, et cetera, which were also responsible for the symptoms for which they sought relief.

Ninety-one persons with convergence insufficiency were given home treatment consisting mainly of the use of a stereoscope and Delta base-out cards. Simple fusion cards made at the office were given children for home use. Other training included the use of loose prisms, base out, finger to nose convergence exercises, and voluntary convergence exercises with finger and distant object (fix on finger at near, being conscious of a doubled distant object, then remove finger and try to keep distant object doubled by looking at imaginary near point).

It can be seen from the foregoing remarks that we have found the Memphis Orthoptic Clinic a valuable adjunct in the handling of our private patients and that we expect to profit by its continued aid in the future.

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DISCUSSION

F. Bruce Fralick, M.D., Ann Arbor, Mich.: A discussion on the value of an orthoptic clinic in private practice is of utmost significance to those contemplating such an organization. Unfortunately, this service now is obtainable in only a few of the larger cities or larger clinics and very rarely in individual private offices. Thus, at present, and for a long time to come, only a very small proportion of individuals requiring orthoptic training will be privileged to receive an adequate course of such exercises.

There are at the present time only sixty-nine technicians licensed by the American Orthoptic Council. Technician training centers are limited and the small number of individuals taking up this work in no measure supplies the demand. Such a demand, however, should attract more and more technicians into this field and so increase the availability of this training to the private practitioner.

It has been clearly demonstrated in the discussion that an orthoptic clinic has been of immense value to Dr. Ellett and his associates. A pooling of common interests and efforts into a cooperative orthoptic clinic undoubtedly has, in a large measure, contributed to the success of the Memphis Orthoptic Clinic. Their ability to subsidize the clinic and thus remove any resemblance to commercialism by not asking fees for the exercises has displaced one of the largest stumbling blocks in the successful management of similar clinics elsewhere.

They have made no radical claims for the cure of cross eyes by orthoptics. They have carefully pointed out that orthoptics is just one of the associated methods in the treatment of strabismus. They also acknowledge that orthoptic training alone can do little, but when intelligently associated with refraction and surgery can, in some instances, accomplish results unobtainable if such training were not utilized. Their statistics obtained through the use of appropriate orthoptic exercises in conjunction with adequate refraction and investigation of the patient's general medical status, revealed gratifying results in the management of non-surgical cases of muscle imbalance and fusional anomalies.

An orthoptic clinic is valuable in private practice if the services can be made available inexpensively and without too great an effort on the part of the patient or parent. Only in this way will a fairly high percentage of the candidates continue treatments long enough to enable one to sort out the suitable cases for training. The clinic must work in close cooperation with the ophthalmologist. Orthoptic technicians should not be expected to correct the symptoms or complaints of all dissatisfied refraction cases. We have found that the orthoptic clinic is often called upon to perform miracles in correcting some of the disturbances of ocular motility which should have been found unsuitable for orthoptics in the physician's office. This has tended to place an unwarranted burden and responsibility upon the technician.

My experience in the value of an orthoptic clinic in private practice conforms closely to that of Dr. Ellett and his associates. Unlike the Memphis Clinic, our clinic is in a city of only 35,000 people, giving rise to certain difficulties we have as yet not surmounted. These difficulties greatly lessen the usefulness of the clinic to the practicing physician. Because the majority of you likewise practice in smaller communities, our experience may be of some help to you in overcoming these same objections.

Our clinic employs the only licensed orthoptic technician in the state of Michigan. These services are available to all the opthalmologists in Michigan. Although the clinic is located in the University Hospital, we have not been able to subsidize the clinic, making is necessary for it to pay its own way. The frequency of the treatments, and the necessity of charging a small fee of approximately two dollars for each treatment without noticeable improvement by parents of squinting children has caused many to discontinue treatment. We have found adults with convergence, divergence, or fusional anomalies more cooperative because they generally appreciate the benefits much sooner.

The second stumbling block in our organization is due to the fact that eighty-five per cent of our patients come from 80 to 800 miles for their medical care. Because of these great distances only a small number can be induced to expend the necessary time and expense of travel to and from the clinic. For this same reason few physicians, other than those in Ann Arbor or in towns relatively close, have utilized the services of the clinic. From this point of view, we can safely say that our own orthoptic clinic has been of little direct value to the ophthalmologists of Michigan. This obstacle can be overcome only by greatly increasing the number of such clinics throughout the state.

The ophthalmologists practicing in Ann Arbor have found our orthoptic clinic of great value in overcoming amblyopia, the determination and correction of abnormal retinal correspondence, central suppression, and fusional and motor anomalies. Although preoperative orthoptic treatment to break up ab-

normal retinal correspondence and to secure functional results is of great importance, we have had a few cases where preoperative treatment could not be given. In a few such cases, we were surprised to find by postoperative synoptophore examinations that normal retinal correspondence developed where the eyes were made straight or nearly straight by operation. It is my belief, therefore, that we cannot lay all our failures to keep eyes parallel after operation to a faulty retinal correspondence. I am also sure that the choice and time of operation plays a large role in one's success or failure.

A careful analysis and comparison of the pre- and postoperative findings in those squinting individuals not able to receive pre- or postoperative orthoptic exercises should be of great interest in further estimating the value of orthoptic clinics in private practice.

OSCAR WILKINSON, M.D., Washington, D. C.: I wish to state that I have followed the development of orthoptics from the day of Claude Worth when he used to take the little child on his lap and work with a small lamp, his first model of the amblyoscope. I have seen him spend a whole half hour of his time—his valuable time—with one child. Of course, with our modern technics in orthoptics, and with better instruments, we are going to get better results.

Orthoptics has passed through different stages. At times it was in bad repute. Then there was a period of endurance, and today it is going to be accepted as a very definite beneficial aid to the cross-eyed child. As I see the orthoptic clinic, it has several valuable features. In the first place, by giving better service, there would be less tendency for the optometrist to waste a lot of the patients' valuable time on these cases. I have just recently had a patient who had been under the care of an optometrist for two years, visiting his place twice a week and, of course, without any particular benefit. If we had clinics of the type that Dr. Ellett and his co-workers have established in different places, I think the public would soon realize that it is a perfect waste of time to patronize such unprepared persons as most optometrists.

The chief function, as I see it, from the orthoptic standpoint is *diagnosis* and *prognosis*. A child comes into our office with a crossed eye. The history is taken and the amount of visual acuity noted, as well as the duration and degree of the squint; then we send the patient to the orthoptist and he is studied for his visual acuity, his fusion ability, his correspondence, whether abnormal or normal retinal correspondence. A patient of any reasonable age, with a normal correspondence, with ability to get secondary fusion, is always a hopeful case. If it is a case of abnormal retinal correspondence and amblyopia of long duration, the prognosis in that case is always bad and we should always tell the parents of these children. The orthoptic department can aid us very decidedly in determining which patient is ultimately going to be relieved and which will not.

The doctors have brought out the fact very well that those older patients with inability to read comfortably are often doing too much work and have an insufficiency of convergence, particularly for near work. Proper orthoptic training gives the quickest relief.

Just one more remark, and that is with reference to the general condition of the patient. This is reflected in the condition of the eye, and the patient in whom the amount of deviation varies from day to day should be carefully examined by the general practitioner for some kind of deficiency in general health.

SOME USEFUL PLASTIC PROCEDURES IN OPHTHALMOLOGY

GEORGE WARREN PIERCE, M.D.
SAN FRANCISCO, CALIFORNIA

I APPRECIATE the privilege of addressing this assembly on plastic surgery about the eye. You will understand my hesitancy in invading the field of the ophthalmologist, whose knowledge of that anatomically small field is thorough. Perhaps a correlation of that knowledge with the experience of the plastic surgeon in the planning and management of tissue transplantation will lead to better results for the patient. This paper presents a limited number of cases illustrating some of the procedures of plastic surgery about the eye.

There is no excuse for the performance of plastic surgery about the eye unless function and appearance are restored. The procedure must be planned carefully. First in planning comes the correct estimation of tissue loss, or of the defect to be filled. Scar tissue contracts, and often when an apparently small scar is completely excised, an appalling defect results, for which a previously cut skin flap will be quite inadequate. Only experience arms the surgeon against this too common mistake. Only experience teaches the best selection of materials for repair of the part.

These statements can be illustrated best by a concrete example.

Case I. — This patient, when ten years old, stood watching a steam roller in the street. The boiler exploded and his face was frightfully burned. Twenty-five years later he came seeking relief from his facial deformities. Extreme ectropion of the right lower lid demanded first attention.

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The skin of the eyelids differs from the skin of any other part of the body. It is very thin and soft and possesses almost no subcutaneous fat, and folds easily. There is a very sharp line of demarcation, quite accurately marked by the bony orbital margin. All skin beyond this margin is much thicker, possesses a heavier fat and fibrous subcutaneous fascia and will not fold easily. This is of the greatest importance in selecting a method of repair of the eyelids, as such skin will not function in the opening and closing of the lids. In the literature a multitude of procedures for repair of the lids with flaps from the temporal region or face are illustrated with convincing diagrams, but the use of this outside tissue will, while actually filling

FIG. I, a

FIG. I, b

the defect, fail miserably both functionally and cosmetically.

We have found that the best material for transplantation to an eyelid defect is eyelid skin. As there is usually a considerable amount of redundant skin of the upper lid, part of this can be borrowed for the lower lid defects if they are small or moderate in size. The skin may be used as a pedicled flap or a free graft, but if the defect is large other material must be found. The skin of the posterior surface of the external ear is thin and inner side of the upper arm is next in order of choice.

In this case the skin behind the ear was selected, as eyelid skin was not available and the defect was foreseen to be large. In all cases of severe, or even moderate, ectropion it is well to create point adhesions between the lids to be maintained until danger of contracture of the graft has passed.

Fig. I, a. — Scar tissue which caused the ectropion must be thoroughly excised until the lid falls easily into the

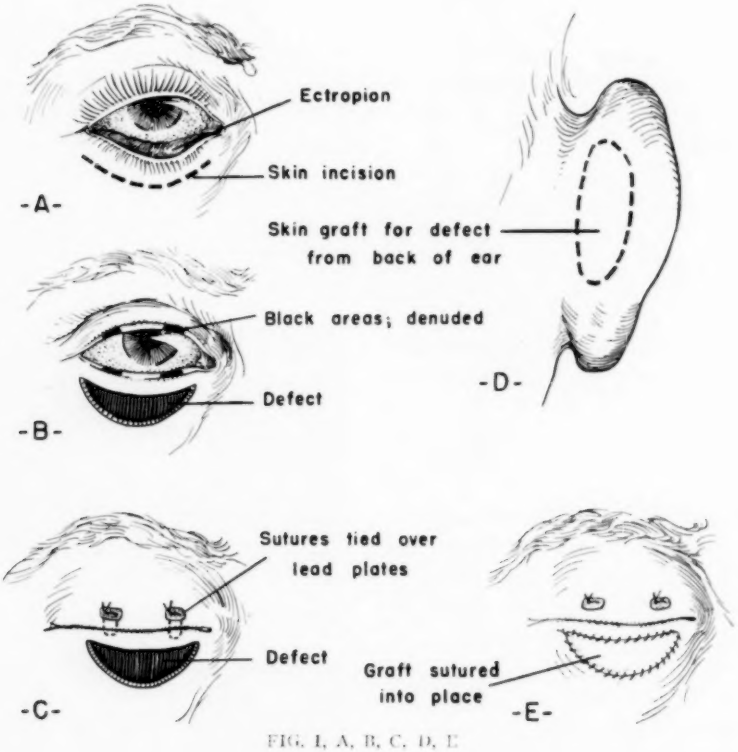

FIG. I, A, B, C, D, E

possessed of very little subcutaneous fat. When used as a free graft it takes easily, matches well in color and texture, and functions efficiently. When even this skin is not available as in many cases of severe burns of the face and head, a thin Thiersch graft taken from the hairless normal position. All free full thickness skin grafts must be carefully splinted, using saline moistened gauze of many thicknesses over the graft, covered with a cotton pad. Finally, Elastoplast, the commonly used elastic adhesive, is invaluable for maintaining the gentle con-

stant pressure on the graft essential for a complete take. The final result is shown in fig. I, b. The surgical procedure is illustrated in fig. I, A, B, C, D, E.

Case II. — This patient suffered a severe burn of the conjunctiva of the left eye several years ago. (Fig. II, a.) This was followed by pericorneal ulceration, which in turn was complicated by a severe infection of the orbit. When the infection was finally cleared the patient was left with a severe diplopia and extreme ptosis. Dr. Roderick O'Connor did several muscle operations with an excellent final result, as the diplopia was relieved. He also performed a Motais operation for the ptosis with a good result but some time later the patient bumped the eye and the ptosis immediately recurred. Because of the massive scarring within the orbit he did not wish to attempt a reoperation of that type and referred her to me. There remained only some type of operation using the frontalis muscle.

Of all types of operations using the frontalis muscle to raise the eyelids we have found transplants of fascia lata strips to be the most satisfactory. The fascia lata is composed of strong longitudinal fibers which have no elasticity and is therefor ideal for transmitting motion from the frontalis muscle to the eyelid. Two tunnels were made from the upper tarsal border to the frontalis muscle; the fascia lata strips were passed through and sutured with 6-0 Anacap silk suture. (Fig. II, A, B.) The functional result is shown in Fig. II, b.

FIG. II, a

FIG. II, b

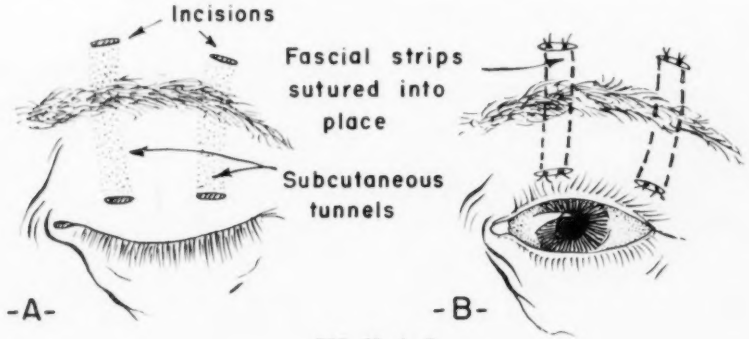

FIG. II, A, B

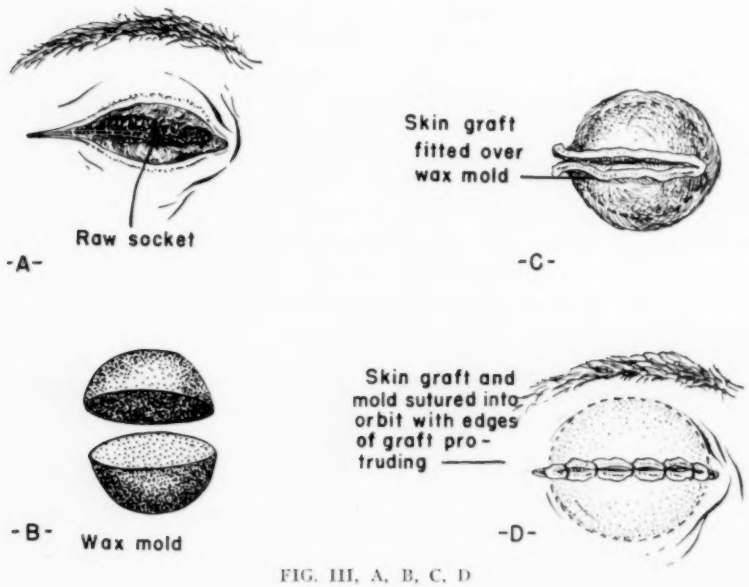

FIG. III, A, B, C, D

Case III. — This patient suffered a lime burn of the right eye at the age of nine years, and the eye was removed. (Fig. III, a, b.) He was seen first by us when an adult. The orbital space was almost obliterated, only sufficient room remaining to insert a navy bean. The entire socket in its anterior half required dissection and skin grafting. The principle of Esser's epithelial inlay was used, after the manner of Dr. John M. Wheeler, whose monumental work on plastic procedures about the eye, with his fine attention to detail and with all elements of his operations satisfying the anatomical and physiological requirements of these problems, has never been surpassed. The great amount of scar tissue present was dissected out, carrying the dissection to the full circumference of the orbit, save in the region of the levator palpabrae muscle. As Wheeler pointed out, this must be done to assure minimum contracture of the graft later, as grafts not anchored to the region of the bony orbit, but only to areolar tissue or mobile tissue tend to contract greatly, with disappointing results. After dissection of the scar a model of the defect was made in two halves (to facilitate its removal). (Fig. III, A, B, C, D.) The model was then wrapped in a single thin Thiersch graft,

FIG. III, a

FIG. III, b

raw surface out, and this was slipped into the cavity, an external canthotomy making this maneuver easy. The lids, including the free edges of the skin graft were sutured together with silk, and a light pressure bandage applied. Ten days later the sutures were removed and the wax model taken out. The graft took completely, as is to be expected when this method is carried out in proper detail. A prosthesis of dental modelling

FIG. III, c

FIG. III, d

wax was immediately modelled and inserted, this to be worn several weeks until all tendency to contracture has passed.

Fig. III, c, shows the interior of the grafted socket two months after operation. The small dark spot at two o'clock in the circumference of the orbit, is mucous membrane at the site of the openings of the ducts of the tear glands. Though the graft covers these ducts completely at the time of operation they always break through the graft within a few days and the mucous membrane grows out from the lining cells of the ducts for a short distance. The final result with an artificial eye is shown in Fig. III, d.

Case IV shows a similar problem which was solved in a different manner. (Fig. IV, a.) This girl lost her eye when twenty-two months old when a fork was stuck into it. She came to us when eleven

FIG. IV, a

FIG. IV, b

years old. Examination showed no inferior sulcus, the region being a bulging soft mass which prevented the wearing of any prosthesis. There was an abundance of normal conjunctiva, so at operation the conjunctiva was incised and a large amount of soft fibrous tissue was dissected out down to the lower border of the orbit. Mattress sutures of silk were then passed through from the cheek, catching the under surface of the conjunctiva at the apex of the cul-de-sac,

and these sutures were tied over a bolster on the cheek. The result was very satisfactory; a deep conjunctival lined sulcus was provided (Fig. IV b) and the patient wearing her artificial eye is shown in fig. IV, c.

FIG. IV, c

Case V.—Correction of entropion has always been difficult, and even if corrected the condition has a marked tendency to recur. The procedure shown in fig. V, A, B, C, D has given for us the most satisfactory and lasting results. As can be seen from the drawings a double pedicled skin flap from the opposing lid was used and let into an incision carefully made between the eyelash line and the actual lid margin. Here it was sutured with 6-0 Anacap suture (Davis and Geck). This silk suture is mounted on an atraumatic needle which is the finest needle for meticulous work that we have been able to obtain. The spear of the needle is so correctly designed that it slips through the tissue with very little pressure and the least amount of trauma. Any needle requiring pressure for passing would tear in two such a small flap. Note also the use of the small hook used in handling the flap. In our work all skin, whether flaps or not, is handled with small hooks. No skin, unless it is to be excised, should be handled with thumb forceps, as the trauma is a definite factor in lowering the vitality of the tissue.

After healing, the pedicles of the flap were cut off and the areas sutured smoothly. This method gives a permanent cure of the entropion unless the pathological condition which originally caused it is progressive.

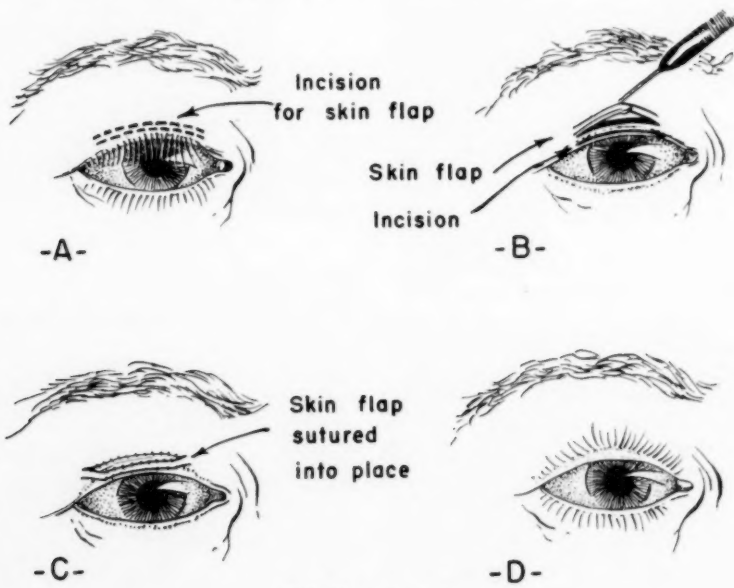

FIG. V, A, B, C, D

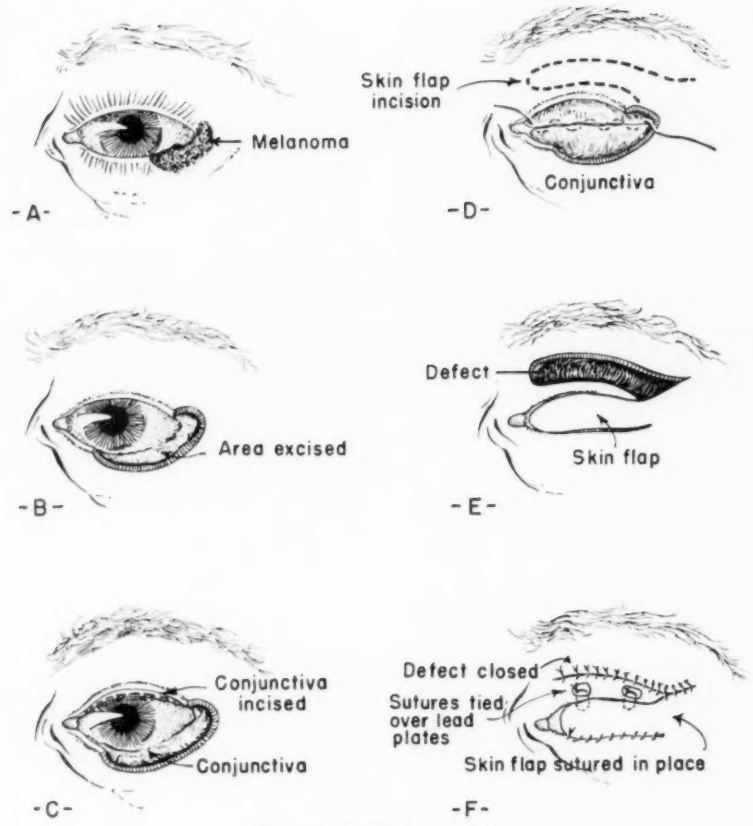

FIG. VI, A, B, C, D, E, F

Case VI. — Melanomas of the eyelid are fairly common. No other skin tumor varies as much in morphology or in degrees of malignancy. Some of the most malignant tumors are melanomas. Excision must be wide and complete. If metastases have not occurred already this excision constitutes a cure. It is our custom at the time of such operations to have an immediate frozen section of the tumor made and examined. Thus complete extirpation of the growth can be ascertained with a fair degree of accuracy. If any tumor cells are seen near the periphery more tissue is removed, the removed growth having been marked before excision so that further removal is done only at the suspected area.

The pigmented growth on this patient's eyelid began to spread four years previously and showed a raised-up active looking papule at its center on the lid margin. (Fig. VI, a.) It extended around the external canthus about 3 mm. onto the upper lid. At operation the entire thickness of the lid was removed, the line of incision extending 5 mm. outside the visible tumor margin. In our opinion repair of such resulting defects should be immediate as the eyeball will thus be protected and scar tissue will be kept at the minimum. Epithelial lining and epithelial covering are both required for this repair. Conjunctiva is considerably elastic and is the ideal tissue for the lining, hence the upper lid was split and

FIG. VI, a

FIG. VI, b

the conjunctival portion sutured to the free cut edge of the lower conjunctiva. Attention should be called to a point of technic of this step. Fig. VI, D, illustrates the manner of suture of the conjunctiva, using a subconjunctival stitch, not passing through the conjunctiva at any point and brought out through the skin at each end. The material was Dermalon 6-0 (drawn, not twisted nylon), Davis and Geck. In this manner no suture material touches the ocular conjunctiva or cornea, avoiding irritation and does not need to be removed for the ten days necessary for firm healing. After the conjunctiva was sutured a transposed flap of skin was outlined on the upper lid, and the flap pedicled just lateral to the external canthus. Here we had ideal material for eyelid outer covering and the flap was transposed downward. Skin suture was done with 6-0

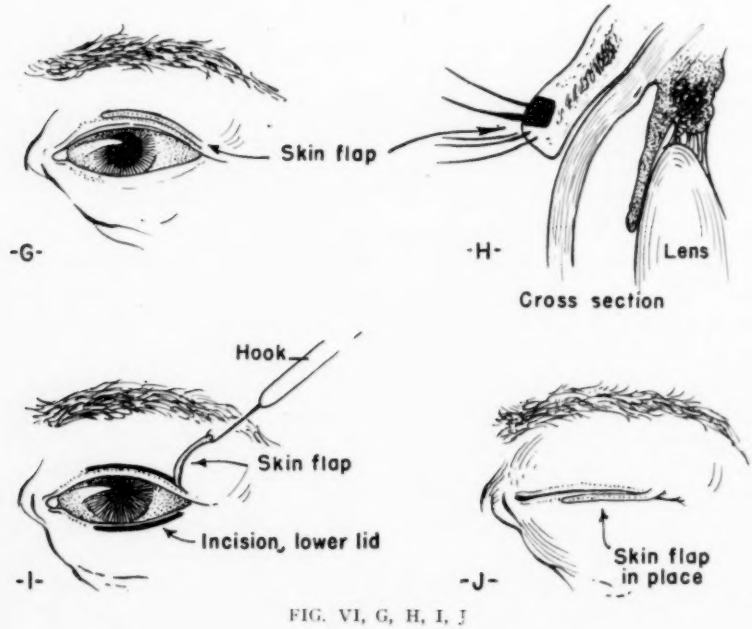

FIG. VI, G, H, I, J

Anacap, and two spots were rawed on the upper lid edge and mattress sutures of 6-0 Dermalon were passed through them and into the upper border of the flap, and were tied over lead plates. The upper evelid defect closed easily. (Fig. VI, D, E, and F.

FIG. VI, c

For two months the tissues remained as sutured (fig. VI, b) to allow for tissue reaction to subside, and then the lids were separated by cutting through with a tenotomy scissors. A functioning lid had been provided and lacked only eyelashes. These were provided by cutting a very narrow pedicled flap from the upper lid, including approximately half of the row of eyelashes, and letting this flap into an incision in the lower lid margin. This incision in the lower lid was placed and angled so that the transplanted upper lid lashes would not curl into the eye. (Fig. VI, G, H, I and J, and fig. VI, c.) The pedicle of this flap was cut only two weeks ago so that final picture cannot be shown.

Case VII (Fig. VII, a) is a patient with the same pathologic finding as shown in case 6, a malignant melanoma. The defect following excision was repaired by the same plan as shown in fig. VI. The initial excision removed more than half the width of the full thickness of the eyelid. Fig. VII, b, shows the result two years after operation. A slight droop of the outer third of the reconstructed lid may be noted. This is due to the absence of muscle in the lid in the rebuilt area.

FIG. VII, a

FIG. VII, b

Fig. VIII a, shows a patient injured by gunshot. The bullet entered above the left supraorbital ridge, ranged downward and backward into the orbit, resulting in complete exenteration. When

FIG. VIII, a

first seen by us the orbital walls in greater part were covered with granulations and the lids were adherent to the scarred area.

The first step of repair was the grafting of granulating areas in the orbit and of the posterior surface of the eyelids after they had been dissected free. This was accomplished by the epithelial inlay method, splinting the thin Thiersch graft with dental modelling wax. The skin grafted orbit is shown in fig. VIII, b,

FIG. VIII, b

and the patient with prosthesis in fig. VIII, c. We are fortunate to have at all times the services of an artificial eye maker who can make eyes to fit any unusually shaped orbit. The prosthesis constructed for this patient is shown in fig. VIII, B. It consists of two parts, a plug with anterior concave surface and an artificial eye with a posterior stern to fit the plug surface.

Obvious in fig. VIII, c, is the sunken appearance of the upper lid below the brow. This deformity is very common in the wearing of artificial eyes and

FIG. VIII, c

"gives away" the wearer. We have tried numerous types of material to correct this. Fat grafts give a fine immediate correction but they absorb rapidly. We now use, as in this case, a dermograft to fill in. Dermografts are prepared by cutting a full thickness, properly patterned, piece of skin from the abdomen and

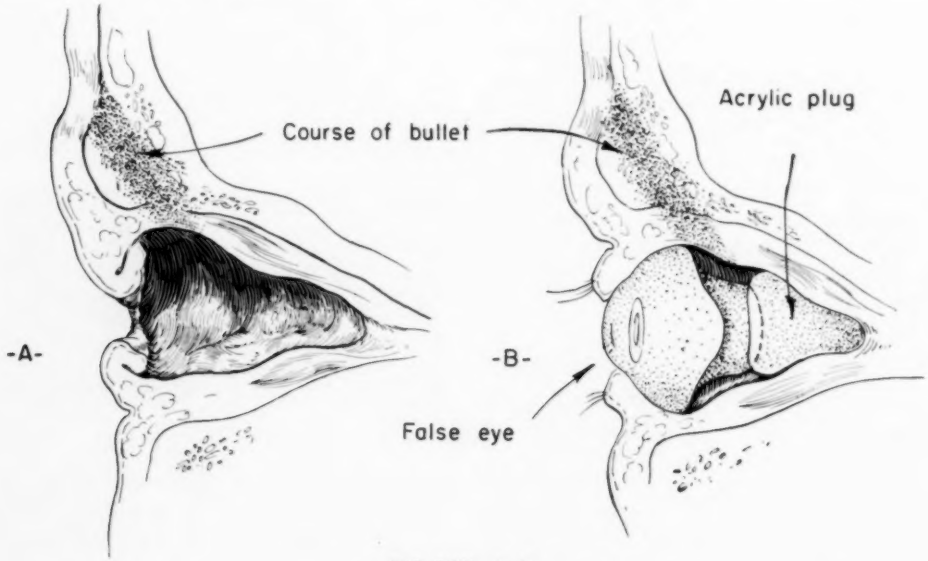

FIG. VIII, A, B

then scraping off the epithelial layer. The area to be grafted is tunnelled through a small incision. A double armed silk suture is passed through the tip of the graft, the needles passed through the tunnel and brought out through the skin at the opposite end of the tunnel. The graft is drawn into its bed and the silk suture tied to maintain the graft in place. The small incision is closed appropriately. A warning should be given not to place the dermograft too close beneath the thin skin of the eyelid. The graft shapes itself to the tunnel and does not absorb. The result of the implantation of such a graft is shown in fig. VIII, d. The final result for this

FIG. VIII, d

patient is considerably less than perfect, but the patient is happy with it and greatly prefers it to wearing a patch or a ground glass lens.

Fig. IX shows a patient whose airplane went out of control at fifteen hundred feet. The plane crashed, his safety belt broke and he was thrown forward violently, his face striking the cowl. His flying goggles were imbedded deeply into his face and right eye. He suffered a skull fracture and a crushing fracture of the right malar bone, the floor of the orbit and all of the bones of the nose. Following the accident his general condition was so precarious that nothing was done to elevate the malar bone and the floor of the orbit. At the time of the accident the eye was ruptured and the entire iris was lost.

We first saw him several months after the accident. Ever since his injury he had suffered from a marked diplopia, present in every direction. As can be noted in fig. IX, a, the right eye is much lower than the left and it is sunken. His discomfort from the diplopia suggested an attempt to ameliorate this condition by surgery. It was evident that the globe of the eye must be raised and brought forward, raised to place the eyes on approximately the same plane, and brought forward to restore the normal pull of the intraocular muscles, so that synergy would be reestablished. In our opinion

FIG. IX, a

the best material for implantation is preserved refrigerated costal cartilage. These isografts are preserved in a solution of aqueous merthiolate one part and normal saline solution four parts, and are kept in a refrigerator. We have used this material for the repair of defects for the past ten years and have made several hundred implantations.[1] We have found that it is superior in many ways to autografts and its use saves the patient the very painful operation of costal cartilage removal.

At operation an incision was made along the lower border of the orbit. The periosteum of the floor of the orbit was incised and elevated with a septal elevator. (Fig. IX, B.) A brain spoon was in-

FIG. IX, b

serted beneath the periosteum and the eyeball elevated until the patient stated that the diplopia was relieved in large part. The space below the eyeball was gauged and the piece of cartilage was shaped to fit. After inserting the cartilage the periosteum was closed with plain catgut and the skin was sutured. (Fig. IX, C, D, E.) Immediately after the operation the patient stated that the diplopia was in large part, though not entirely, relieved. After four weeks the diplopia was entirely relieved for all ordinary use of the eyes, only a slight diplopia recurring when the patient looked far to the right.

The patient's corrected vision is now 20/40 and it is proposed to make a contact lens for him with an artificial iris blown in to protect the retina from bright light.

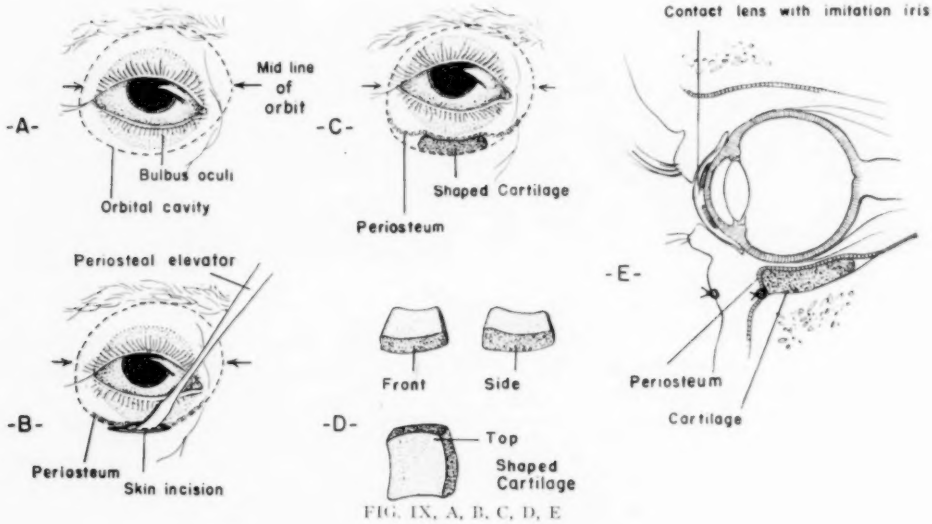

FIG. IX, A, B, C, D, E

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DISCUSSION

HAROLD F. WHALMAN, M.D., Los Angeles, California: The axiom, "Necessity is the mother of invention," finds frequent fulfilment in the sphere of surgery and perhaps particularly in the field of reconstructive surgery where conditions are constantly at variance and ingenuity takes precedence over established procedures to meet the requirement of each case.

Dr. Pierce's demonstrations of the lining of the orbit with skin grafts were excellent, particularly the one giving the prothesis additional support by means of implanted plastic material. The cartilage implant for support of a sunken eyeball gives us a chance to take care of cases that we have never before been able to restore to any decent looking appearance.

With reference to the use of skin about the eyes, there is an axiom that nothing is better to replace skin of the eyelids than skin from other eyelids, in other words, making use of skin flaps from the same side or from the opposite eyelid in the manner which John Wheeler did so expertly.

The report of a newly devised procedure in no way disparages the value of previously reported methods and if it solves a particular problem it would seem odious to review all other operations, discuss and rationalize them in order to justify the usefulness of another.

I wish to present as a preliminary report a new procedure developed out of the necessity for extensive ablation of the right lower lid for a basal cell carcinoma which had grown in both directions laterally from the middle of the lower lid and had invaded the tarsal cartilage. The growth was invasive in character, did not subside with radiation, and since larger dosage would result in lid destruction, excision with a safe margin beyond its limits seemed advisable.

The lower lid was therefore excised by sharp dissection beginning at the external canthus and making a curvilinear incision through the skin down along the inferior orbital margin to about 3 millimeters nasalward of the punctum. The tarsal plate and conjunctiva were then included in the excision by using scissors to complete the ablation. The lower canaliculus was split for drainage.

The first step after ablation was to prepare a broad pedicle skin flap from the upper lid in the following manner. A curvilinear incision was made in the skin including the orbicularis in a natural crease about 5 millimeters distal to the upper lid margin. This was then carefully undermined just beneath the orbicularis for a distance of 15 millimeters to make a skin-orbicularis flap.

The second step of the operation was to return to the lower cul-de-sac where the conjunctiva was undermined down to the fornix. There is considerable conjunctiva left in the fornix even after removing the lid and furthermore it can be stretched considerably after it is undermined. Into the conjunctival flap four double armed black silk sutures were inserted from the conjunctival side and each in turn then inserted through the orbicularis-skin flap coming out in the skin. These were tied over small pieces of rubber on the skin side giving the skin-orbicularis flap a conjunctival lining and of course the eyeball was then obscured. The free margin of the skin-orbicularis flap was then united to the skin-orbicularis edge of the incision made through the lower lid by means of closely placed interrupted black silk sutures.

Union was allowed to take place for ten days and then all sutures were removed and the upper lid flap was severed from its base. The wound margins of the upper lid were trimmed and united leaving a cleanly united upper lid from which a skin flap had replaced the ablated skin of the lower lid.

The new lower lid was actually larger than needed, but to allow for shrinkage the excess was not trimmed. The skin and conjunctiva were united for smoothness however.

Two months later little shrinkage had taken place so the lower lid was reduced to better size by excising some of it at its margin.

The lower canaliculus which had been split remained patent and because the lower lid of the fellow eye had imperceptible eyelashes no attempt was made to implant hair-bearing skin for lashes. It is my opinion that they are usually so inconspicuous on the lower lid that the procedure is not always necessary for good appearance.

Exceedingly good function and appearance have resulted from the operation described. The lid is fully reconstructed with the exception of cartilage which does not seem to be important in the lower lid and the finished work is smooth and even with no perceptible scars as union takes place in the natural folds of the upper as well as the lower lids. The procedure is obviously not suited to reconstruction of the upper lid.

GEORGE W. PIERCE, M.D.: I want to thank Dr. Whalman for his discussion, and I want to say one thing about the last case I showed. That eye was not only lifted up but brought forward, increasing the normal pull on the muscles. That can be done by properly shaping the piece of cartilage. Dr. Whalman has a very nice operation. Both are about the same in principle, and I will say this one thing about skin flaps, and that is that circulation in them is important, particularly from the point of view of the venous return. It is better to have a flap that is pale than a flap that is cyanotic, because cyanosis means

FIG. IX, b

serted beneath the periosteum and the eyeball elevated until the patient stated that the diplopia was relieved in large part. The space below the eyeball was gauged and the piece of cartilage was shaped to fit. After inserting the cartilage the periosteum was closed with plain catgut and the skin was sutured. (Fig. IX, C, D, E.) Immediately after the operation the patient stated that the diplopia was in large part, though not entirely, relieved. After four weeks the diplopia was entirely relieved for all ordinary use of the eyes, only a slight diplopia recurring when the patient looked far to the right.

The patient's corrected vision is now 20/40 and it is proposed to make a contact lens for him with an artificial iris blown in to protect the retina from bright light.

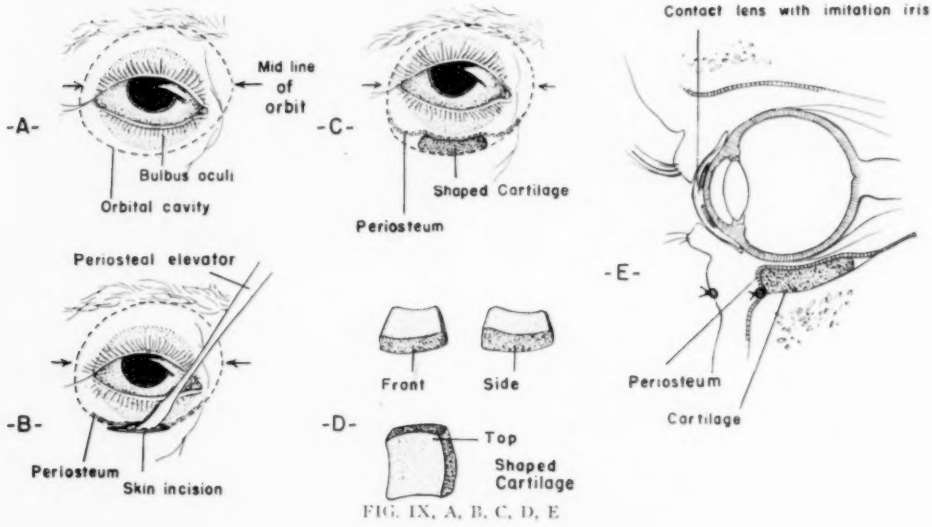

FIG. IX, A, B, C, D, E

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DISCUSSION

HAROLD F. WHALMAN, M.D., Los Angeles, California: The axiom, "Necessity is the mother of invention," finds frequent fulfilment in the sphere of surgery and perhaps particularly in the field of reconstructive surgery where conditions are constantly at variance and ingenuity takes precedence over established procedures to meet the requirement of each case.

Dr. Pierce's demonstrations of the lining of the orbit with skin grafts were excellent, particularly the one giving the prothesis additional support by means of implanted plastic material. The cartilage implant for support of a sunken eyeball gives us a chance to take care of cases that we have never before been able to restore to any decent looking appearance.

With reference to the use of skin about the eyes, there is an axiom that nothing is better to replace skin of the eyelids than skin from other eyelids, in other words, making use of skin flaps from the same side or from the opposite eyelid in the manner which John Wheeler did so expertly.

The report of a newly devised procedure in no way disparages the value of previously reported methods and if it solves a particular problem it would seem odious to review all other operations, discuss and rationalize them in order to justify the usefulness of another.

I wish to present as a preliminary report a new procedure developed out of the necessity for extensive ablation of the right lower lid for a basal cell carcinoma which had grown in both directions laterally from the middle of the lower lid and had invaded the tarsal cartilage. The growth was invasive in character, did not subside with radiation, and since larger dosage would result in lid destruction, excision with a safe margin beyond its limits seemed advisable.

The lower lid was therefore excised by sharp dissection beginning at the external canthus and making a curvilinear incision through the skin down along the inferior orbital margin to about 3 millimeters nasalward of the punctum. The tarsal plate and conjunctiva were then included in the excision by using scissors to complete the ablation. The lower canaliculus was split for drainage.

The first step after ablation was to prepare a broad pedicle skin flap from the upper lid in the following manner. A curvilinear incision was made in the skin including the orbicularis in a natural crease about 5 millimeters distal to the upper lid margin. This was then carefully undermined just beneath the orbicularis for a distance of 15 millimeters to make a skin-orbicularis flap.

The second step of the operation was to return to the lower cul-de-sac where the conjunctiva was undermined down to the fornix. There is considerable conjunctiva left in the fornix even after removing the lid and furthermore it can be stretched considerably after it is undermined. Into the conjunctival flap four double armed black silk sutures were inserted from the conjunctival side and each in turn then inserted through the orbicularis-skin flap coming out in the skin. These were tied over small pieces of rubber on the skin side giving the skin-orbicularis flap a conjunctival lining and of course the eyeball was then obscured. The free margin of the skin-orbicularis flap was then united to the skin-orbicularis edge of the incision made through the lower lid by means of closely placed interrupted black silk sutures.

Union was allowed to take place for ten days and then all sutures were removed and the upper lid flap was severed from its base. The wound margins of the upper lid were trimmed and united leaving a cleanly united upper lid from which a skin flap had replaced the ablated skin of the lower lid.

The new lower lid was actually larger than needed, but to allow for shrinkage the excess was not trimmed. The skin and conjunctiva were united for smoothness however.

Two months later little shrinkage had taken place so the lower lid was reduced to better size by excising some of it at its margin.

The lower canaliculus which had been split remained patent and because the lower lid of the fellow eye had imperceptible eyelashes no attempt was made to implant hair-bearing skin for lashes. It is my opinion that they are usually so inconspicuous on the lower lid that the procedure is not always necessary for good appearance.

Exceedingly good function and appearance have resulted from the operation described. The lid is fully reconstructed with the exception of cartilage which does not seem to be important in the lower lid and the finished work is smooth and even with no perceptible scars as union takes place in the natural folds of the upper as well as the lower lids. The procedure is obviously not suited to reconstruction of the upper lid.

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that the venous return is not sufficient, and when lymphatic engorgement occurs, plus venostasis, thrombosis follows and the flap is lost. That is why it is best to take double pedicled flaps rather than single pedicled flaps, and it is also well to splint these with saline gauze and light pressure, because that prevents overcongestion and it also makes them act almost as free grafts, giving a double advantage.

THE LARYNGEAL (VOCAL CORD) NODE

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INTRODUCTION. Elimination of all frank neoplasms and specific inflammations of the vocal cords leaves a group of lesions commonly seen by laryngologists and recognized by them under a variety of terms, usually as "polyps," but also as fibromas, singer's and preacher's nodes, angiomas, amyloid tumors, etc. After a study of the 138 cases of such lesions that have accumulated in your Registry of Otolaryngic Pathology at the Army Medical Museum, it seems to us that these clinical diagnoses represent different stages of the same condition and that they can be consolidated under the single term "laryngeal node" or perhaps more specifically "true-cord node." The 33 cases contributed to the Registry by one of us (L. S.) make possible the inclusion in the study of first-hand experience with the clinical aspects, the treatment and the outcome of the lesions. No matter what you may think of this study, we hope that at least you will be impressed with the importance and possibilites of the Registry program which makes possible the accumulation of a significant number of cases in a minimum of time and their convenient concentration for definitive studies. This applies not only to the subject under discussion, but to all other lesions covered by your specialty. The success of the Registry program is entirely dependent on the cooperation of the clinicians in sending in their significant cases.

Presented at the Forty-Eighth Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 10-13, 1943, Chicago, Illinois.

HISTORICAL. We were in the habit of diagnosing these lesions on the basis of their histologic picture — polyps, varix, amyloid tumors, for example. It finally occurred to us that we were simply seeing the various stages of the same process. On looking into the literature we discovered, of course, that ours was not an entirely original idea. As far back as 1895 Chiari[1] expressed somewhat the same notion, and more recently in 1941, Friedberg and Segall.[2] Waldapfel[6] in 1940 intimated as much in his more general study of cord lesions. We are all indebted to Reinke[5] for his detailed study of the anatomy of the true cord that was later confirmed by Hajek.[3]

ANATOMY. Fig. 1 is the true cord stained for elastic tissue from a carcinoma of the larynx. The resulting edema conveniently demonstrates the so-called "Reinke's layer" and the possible limitation of lesions to this layer afforded by the close apposition of the mucosa to the deeper tissues at the striae arcuata superior and inferior. Note particularly the heavy concentration and the intimate application of the elastica over the cord muscle, a barrier to deeper extension of lesions in Reinke's layer. Note also the absence of gland elements in this region, but the presence of a few elastic fibers just beneath the surface squamous epithelium. The blood vessels are nearer to the deep elastica than to the surface and apparently run parallel to the long axis of the cord.

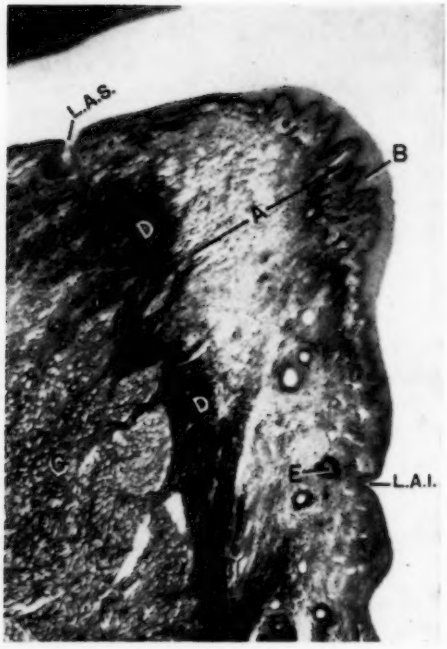

FIG. 1 — Low power of true vocal cord. Elastic tissue stain to demonstrate Reinke's layer (A); Linea arcuata superior (L.A.S.) and inferior (L.A.I.). Hypertrophy of squamous epithelium (B); muscle (C); elastica (D); mucous gland ducts (E). Note distance between elastica and linea as compared with that to free edge of cord.
A.M.M. neg. No. 76559

ETIOLOGY. In practically every one of our cases there is a specific history of misuse or excess use of the voice, ranging from the acute screeching at a football game to the prolonged daily "hup, 2, 3, 4" of the drill sergeant. This experience, together with the scarcity of active inflammatory reaction in the biopsies, has led us to consider trauma as the basic etiologic factor. In only 9 of the cases is there history of noteworthy nasal, pharyngeal or sinus disease. This infrequency would seem to eliminate infection from consideration. In the cases personally observed (L. S.), laryngitis, acute or chronic, was uncommon, and the vocal cords, except for the focal lesion, were generally of normal color and thickness. A small infantile larynx was noted in one young female, and adductor weakness was found frequently among female patients. These might be considered as possible causes of vocal strain. In no instance was the afflicted of the soft-spoken or mild-mannered type, but rather of the garrulous, noisy makeup.

TABLE I

138 Cases (75 Army, 33 L. Schwartz)

Age, Years	*No. Cases*
Under 10	1
10-20	2
20-30	42
30-40	33
40-50	31
50-60	14
60-70	4
Unknown	11
Youngest age	4
Oldest age	64

Sex		*Race*	
Male	114	White	129
Female	22	Colored	5
Unknown	2	Unknown	4

CLINICAL PICTURE. The distribution as to age, sex and color is given in table I. It will be noted that there is a proportion of approximately 5 to 1 in favor of the male. It is true that 75 of our cases are from Army personnel in which, of course, the male sex predominates, and this has influenced the distribution as to sex, but among the other cases in the Registry the majority is still predominantly male. It is obvious that the opportunity for unusual use of the voice is greater with the male. The disproportion between white and colored might be presumed to be based on the roomier, more relaxed and deeper pitched larynx of the Negro. It will be noted that the majority of cases occur in the middle aged when voice strain is more likely.

The universal and primary symptom is hoarseness. This may be acute in the case of sudden voice strain, or of gradual development in those with less severe but sustained over-use of the voice.

In our series there was only one with an acute onset; with the others it was a matter of months to over 5 years before seeking medical advice.

On examination, the appearance of the lesion depends on the stage. As stated above, except for the local swelling, the cords are in the majority of the cases of normal color and size. The great majority of the nodes has been in the anterior third of one or the other true cord. The distribution, in those cases in which it has been recorded for us, is given in fig. 2. This is necessarily diagramatic and those recorded in the anterior commissure and interarytenoid space have been extensions from the anterior ends of the cords and from the artyenoid eminence. An occasion lesion has been of a more diffuse nature, but we have been careful to eliminate any case in which there was question of frank papilloma. In 21 cases the lesions were reported as being multiple.

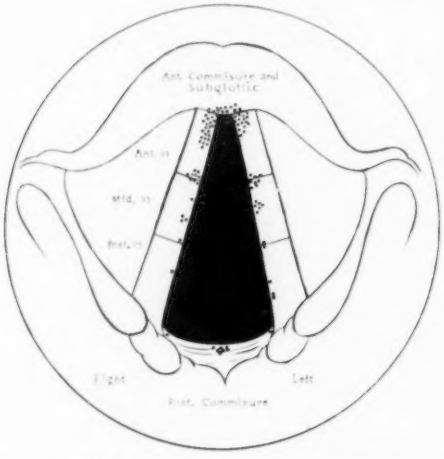

FIG. 2 — Diagram of larynx to show distribution of nodes.

Based on the histopathology we can recognize four types—fibroid, polypoid, varix and hyalin ("amyloid tumor"). The experienced laryngologist can usually differentiate these types clinically. The fibroid is a low, rather firm, pale, not so sharply demarcated node; the polypoid is a sharply defined, more or less pedunculated and translucent mass; the varix more colored from blood content, but not so easily recognized because there is polypoid tissue, and usually more or less hyalin; the hyaline node is pale, quite firm, at times hard.

Ulceration is not common even in the pedunculated nodes.

PATHOLOGY. There is no sharp demarcation between the four stages in the picture, nor is there close correlation between the duration of the lesion and its histology. In table II are tabulated the four types according to duration. This would seem not to substantiate our thesis that the lesion begins as a thickening of the connective tissue element in Reinke's layer, a proliferation of young blood vessels, and the exuding of serum. It will be noted in the table that such a pic-

TABLE II

Duration	*Fibroid*	*Polypoid*	*Varix*	*Hyalin* ("Amyloid")*	*Totals*
Under 1 month	2	2	—	—	4
1-6 months	2	20	3	3	28
6 months-1 year	4	9	1	3	17
1-2 years	3	4	2	2	11
2-3 years	1	13	1	3	18
3-4 years	3	1	—	2	6
4-5 years	1	3	—	1	5
Over 5 years	2	10	2	4	18
Totals	18	62	9	18	107†

* 25 specimens were negative to specific amyloid stain.
† Duration not recorded in 31 cases.

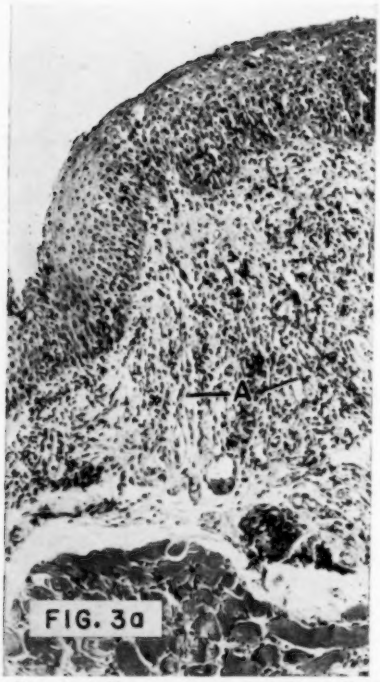

FIG. 3a — Hoarse two weeks. Early fibroid with inflammation. Early proliferation of blood vessels (A) at right angles to long axis of cord.

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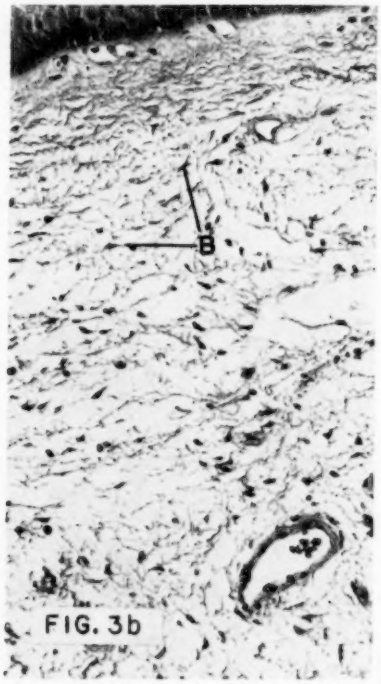

FIG. 3b — Hoarse three years. Polypoid, showing fibroblastic proliferation; edema; young fibroblasts (B).

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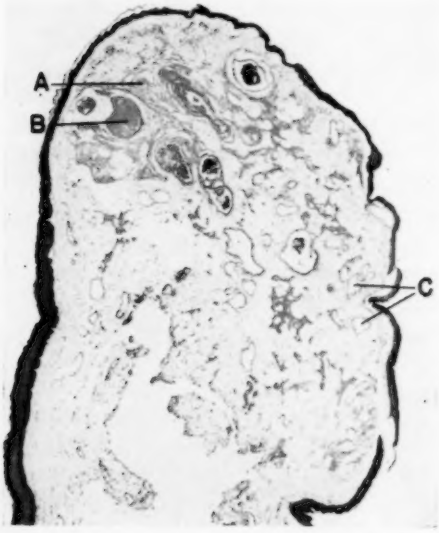

FIG. 4 — Hoarse three years. Polypoid. Early interstitial (A) and vascular (B) hyalin; young blood vessels (C) dilated. Epithelium atrophic.

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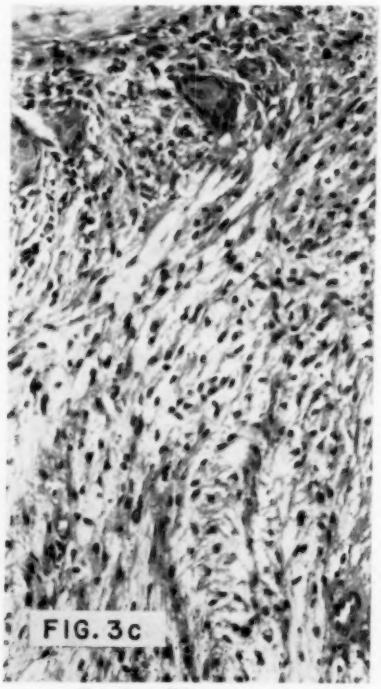

FIG. 3c — Fibroma, neoplastic.

A.M.M. neg. No. 76455

ture was found in cases of over 5 years duration. We interpret these long standing fibroid lesions as still in the primary stage and believe that they have not progressed to the polypoid or hyaline stages because of more favorable anatomic conditions of the larynx and a low grade of trauma. Fig 3a is an example of such a lesion. It shows more inflammatory reaction than is usual, but illustrates very well the change in vascularity — the young blood vessels extending from the deeper original vessels out toward the epithelium surface. There is the possibility that the pathologist sees fewer of the fibroid nodes because the patients do not consult the laryngologist soon enough.

The commonest stage is the polypoid, exemplified in fig. 3b. There is actual proliferation of fibroblasts, but not of the neoplastic type. They are young, stellate, and the result of irritation. These fibroblasts are widely separated by the interstitial edema and the picture is quite comparable to the polypoid reaction in other mucosae. The new blood vessels are scarcely recognizable in their dilated state from the edematous interstices. The scarcity of exudate is noteworthy. Fig. 3c is from a fibroma of the larynx and has been included to em-

FIG. 5 — Hoarse four months. Varix stage. Thrombosis (A); dense interstitial hyalin (B) that has practically eliminated the polypoid feature; epithelium atrophic. Note tortuous, dilated young vessel (C) extending to surface epithelium.
A.M.M. neg. No. 76455

phasize the difference between neoplastic and inflammatory proliferation of the fibroblastic tissue. Fig. 4 is the more usual picture in the polypoid stage and includes the early appearance of hyalin.

We have comparatively few in the varix stage because so many in which the vessels had dilated (telangiectatic) perceptibly were still predominantly

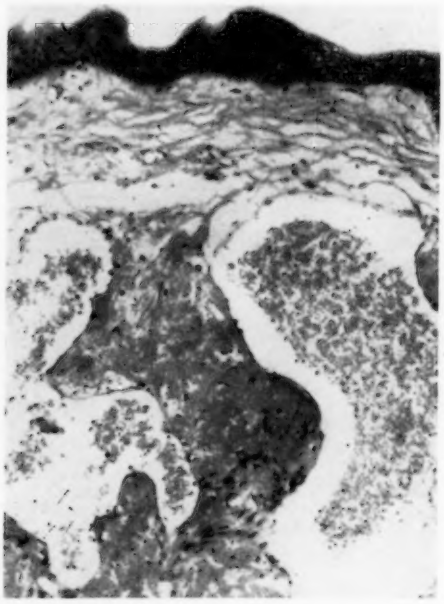

FIG. 6—Hoarse two years; varix; interstitial hyalin. A.M.M. neg. No. 65222

polypoid and were so classified; in others the hyaline element and thrombosis had obliterated the vessels. Figs. 5 and 6 illustrate our varix stage. In both it will be noted that there is considerable interstitial hyalin.

Hyalin is a comparatively late development, but is present in its early phases in the majority of the nodes. It is only when it dominates the picture that we have classed the node as in the hyaline stage and we consider this the final stage. We recognize three distributions. First, interstitial, resulting from fusion and hyalinization of interstitial fibrils; second, the hyalinization of intravascular thrombi; and third, hyalinization of the vascular walls. The three types can usually be found in most of the nodes, but usually one predominates, especially either interstitial or thrombotic. The morphology and presence in the vessel wall suggest amyloid, but in none of the 25 specimens which we stained for amyloid was it demonstrated. Amyloid does occur in the larynx which is occasionally the primary site, but we feel that that is another story and not part of the node picture. Figs. 7a and b illustrate the "amyloid" stage and its vascular and interstitial distribution. Fig. 8 illustrates thrombosis of telangiectatic vessels with beginning and complete hyalinization of the thrombi.

Hemorrhage has not been a common feature in our series and most of the hemorrhages seen have been so recent as to suggest that they are operative. Occasionally, however, older hemorrhages have been encountered evidenced by diffusion of blood pigment and early organization. A recent hemorrhage can be seen in fig. 7a. The infrequency of hemorrhage is rather remarkable when we consider the usual thin-walled vascularity of the nodes.

The epithelium covering the majority of the nodes is usually atrophic, but in a number of instances it is hypertrophic with exaggeration of the rete pegs. Occasionally this feature has reached the degree of pseudo-epitheliomatous hyperplasia, as illustrated in fig. 9b, where the apparent isolation of small groups of epithelial cells suggests malignancy. Experience and careful study are necessary at times to eliminate this possibility. It will be found, however, on serial section, that these apparently isolated nests are actually extensions of rete pegs. In only one instance in our group have we found actual malignancy, illustrated in fig. 9c, an early epidermoid carcinoma. In this connection it is well to

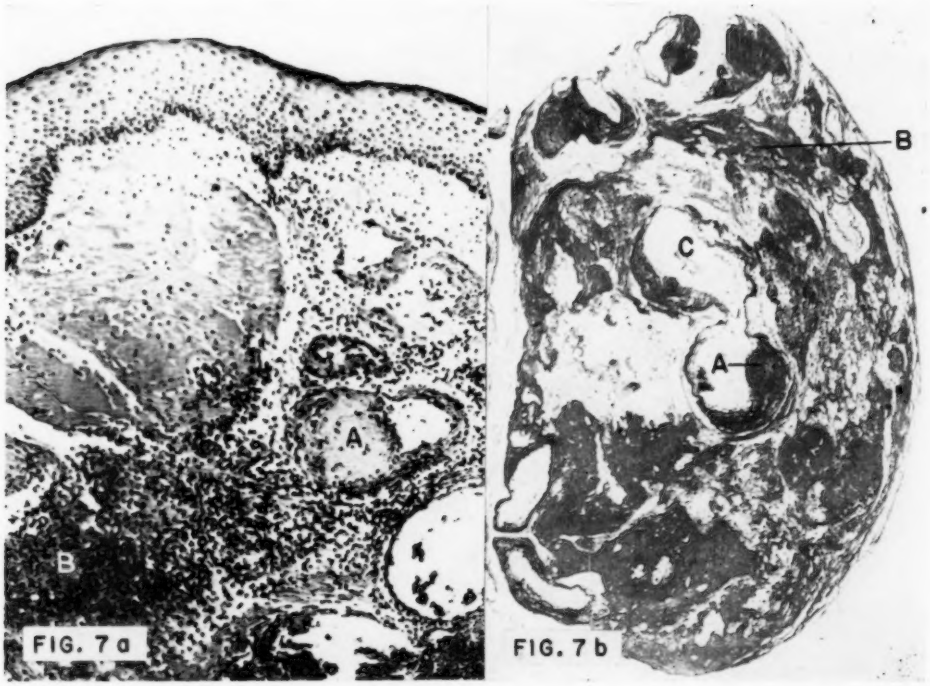

FIG. 7 — Hoarse five months. Hyaline ("amyloid") stage.
a. Vascular hyalin (A); recent hemorrhage (B); moderate epithelial hyperplasia.
A.M.M. neg. No. 76534
b. Vascular hyalin (A); interstitial hyalin (B); telangiectasis (varix) (C).
A.M.M. neg. No. 76460

call attention to Waldapfel's[6] eighth case, a carcinoma of the cord, that was apparently limited to Reinke's layer by the barriers formed by the linea arcuata and the practical absence of lymph vessels, suggesting the possibility of eradication by laryngeal fissure.

Several of the nodes have shown, in addition to hyperplasia of the epithelium, hyperkeratinization-pachydermia to the laryngologist. This is illustrated in fig. 9a.

TREATMENT. As practiced by one of us (L. S.) treatment is along the following lines. The well-recognized means of removal with cup-shaped laryngeal forceps is the only method of treatment worthy of mention. It is best accomplished by direct laryngoscopy. An armamentarium of five such forceps (large and small straight, large and small angulated and Imperatori subglottic), suction apparatus and swabs, has proven adequate for the handling of all such cases. The proximally-lighted Chevalier Jackson type anterior-commissure laryngoscope is preferred, although on occasions the distally-lighted electroscopes with laryngeal spatulae have been used satisfactorily. It goes without saying that one should be prepared, both as to skill and to instrumentarium immediately available, to handle successfully any eventuality that might arise in such operations. Fortunately they are remote and exceedingly rare.

There still are exponents of mirror or indirect laryngoscopy. The direct method has proved itself vastly superior and

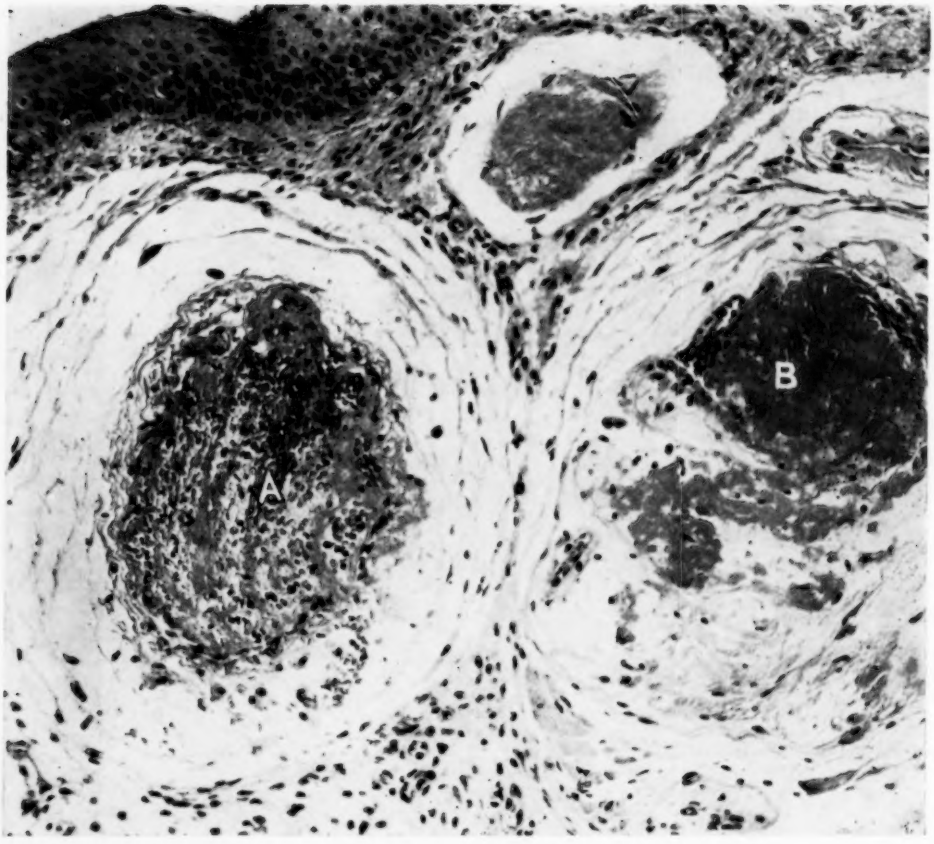

FIG. 8 — Hoarse 3 years. Polypoid. Thrombus, early (A), late (B) with complete hyalinization. A.M.M. neg. No. 76452

completely adequate in all our cases, however. Several cases had had unsuccessful attempts made at removal by indirect laryngoscopy elsewhere. Many of these cases are very difficult to remove by mirror laryngoscopy, particularly those having partial or complete subglottic attachment, whereas they present no problem at all by direct means.

The all-too-frequent attempts at removal by applications of caustic substances to the delicate cord are condemned as ineffectual and dangerous.

PROGNOSIS: The outlook is almost universally good. With complete and meticulous removal there is, as a rule, immediate restoration of the normal voice. Vocal rest should be demanded during the postoperative period of tissue reaction to instrumentation and tissue repair. In those cases of generalized polyposis the "stripping" operation as devised by Lore[4] is advocated. In the 33 cases of L. S. there was one patient who had two recurrences; this was the lone exception to a complete lack of recurrences. In the total group, however, there were recurrences in 12 cases.

CONCLUSIONS: The clinical and pathologic analysis of 138 laryngeal nodes has been made, as a result of which it is our opinion that:

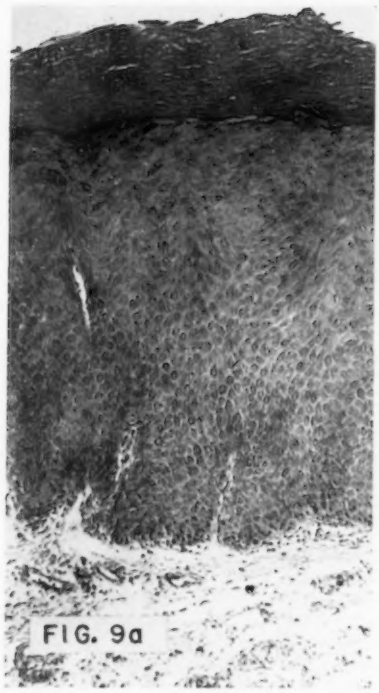

FIG. 9 — Epithelial types.
a. Hoarse six weeks. Hyperkeratosis — "pachydermia" — early polypoid.
A.M.M. neg. No. 82301

FIG. 9 — Epithelial types.
b. Hoarse two years. Pseudo-epitheliomatous hyperplasia. The apparently isolated nests of epithelium are actually extensions of rete pegs. Polypoid stage with telangiectasis.
A.M.M. neg. No. 76510

(1) They are due to trauma;

(2) They occur most commonly in adult white males;

(3) They occur most commonly as single lesions on the anterior third of either true cord;

(4) They can be differentiated clinically and pathologically into four stages (a) fibroid, (b) polypoid, (c) varicose, (d) hyaline;

(5) The lesions are dependent for their location and development on the peculiar anatomy of the free edge of the true cord, Reinke's layer;

(6) They are amenable to surgical cure with some possibility of multiplicity and recurrence and the remote possibility of malignant change in the epithelium;

(7) There is no evidence in this series of amyloid.

We are greatly indebted to Helenor Campbell Wilder, of the Museum staff, for preparing the clinical abstracts and tables and for her aid in preparing the illustrations and the exhibit.

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FIG. 9 — Epithelial types.
c. Hoarse eight months. Early epidermoid carcinoma. Fibroid stage, recurrent.
A.M.M. neg. No. 76456

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DISCUSSION

FRANK J. NOVAK, M.D., Chicago, Illinois: The paper of Col. Ash and Dr. Schwartz is both interesting and important because it is based upon an impressive amount of pathologic material thoroughly prepared and carefully studied. It clarifies the whole picture. The essayists say that the experienced laryngologist can usually differentiate the types. Clinically, however, it is not of extreme importance to make this clinical differentiation. It is of very great importance, however, to differentiate, between these nodes in their various types and the benign and malignant epithelial lesions which occur on the cords, and this is not always easy even for the experienced laryngologist.

This paper is conspicious evidence that the otolaryngologic registry of the Army Medical Museum under Col. Ash's distinguished leadership is paying dividends.

THE DEVELOPMENT OF THE ORGAN OF CORTI IN RELATION TO THE INCEPTION OF HEARING

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The membranous labyrinth of the opossum (Didelphys virginiana) is still in a relatively early stage of development at birth and is entirely non-functional. This opossum is born 13 days after fertilization and completes its development in the brood pouch of the mother. When born most of its organs are very embryonic, but by 50 days post partum it has reached a stage of development approximating that of most new-born mammals (McCrady[18]). The opossum pouch young therefore provides embryonic material easy of access for experimental purposes.

Langworthy[15] noted the earliest sign of vestibular response in pouch young at 41 days, while Larsell, McCrady and Zimmermann[16] reported such responses at about 43 days. Acoustic function does not appear until about a week later. Larsell and McCrady[17] and Larsell, McCrady and Zimmermann reported acoustic responses, in the form of a spasmodic contraction of trunk musculature, in pouch young at 50 days. This was elicited by shrill, sharp whistling, and by sharp clapping of the hands. The first strong response to a note of known vibration rate (E″, 1305 cycles per second) was obtained at 51 days, with slight responses to notes of 1536 and 1024 c.p.s. Responses to the last named notes increased in the 52-day pouch young. At 53 days, middle C (256 c.p.s.) evoked a trunk reflex. At 55 days this note brought forth a pinna reflex in addition to the trunk response, and on subsequent days, pinna or tail reflexes, in addition to trunk movements, were usually observed in response to each of the notes used in the tests. The volume of sound was not measured in these earlier experiments, but it was noted that the small volume produced by a C″ tuning fork (1024 c.p.s.) did not produce response in any of the ages tested, while the same note on the organette, producing much greater volume, elicited responses beginning at 51 days.

Following these observations McCrady, Wever and Bray[19], using a pure tone apparatus, made reflex tests on pouch young of the Mexican opossum (D. marsupialis) ranging in age from 59 to 82 days. They obtained responses between a range of 1000 to 4500 c.p.s. in the 59-day stage, 800 to 6000 c.p.s. at 66 days and 100 to 19,000 c.p.s. at 80 days

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and older, with 300 to 14,000 c.p.s. for one adult tested.

Measurement with the cathode-ray oscillograph showed a much greater range of response. The method consisted of placing the active electrode of the apparatus in contact with the membrane of the round window, which can be exposed by a simple operation in the pouch young, and the indifferent electrode under the skin of the incision. The animal was secured in position so that the auditory meatus was one inch from the end of the tube leading from a loudspeaker which relayed tones of desired pitch and intensity. The electrical effects produced in the cochlea were led from the active electrode through an amplifier to the oscillograph. The range of frequencies producing responses by this method was about 500 to 6000 c.p.s. at 59 days, 300 to 10,000 c.p.s. at 61 days, and 200 to 20,000 c.p.s. at 68 days, indicating a trend toward both lower and higher frequency responses with progressive development. In 1940 McCrady, Wever and Bray[20] reported another series of tests by the electrical method on a litter of 8 pouch young of the Virginia opossum (D. virginiana), ranging in age from 48 to 77 days when tested. Measurements were made on both ears in six of these pouch young and on one ear only in the remaining two, tonal range as well as intensity of sound required being recorded. The results are indicated in the form of graphs in the paper above cited. For convenience of reference in the present report they are represented in tabular form in table 1.

The first experimental cochleae reported by Larsell, McCrady and Zimmermann[16] in 1935 were studied histologically only in a preliminary way. It was pointed out that the cochlea has acquired approximately the two and one-fourth turns, characteristic of the adult, by 16 days after birth. Hair cells and nerve fibers are present as early as 29 day stages, and probably earlier, and the tunnel is present as a narrow cleft in the middle coil and in the distal part of the basal coil at this stage.

The 48 to 77 day pouch young tested by McCrady, Wever and Bray were prepared for histological study, after fixation of the heads with the cochleae in situ. The temporal bone was sectioned, mounted serially and stained with hematoxylin and eosin. These series were supplemented by untested cochleae from corresponding stages of pouch young, obtained from the Wistar Institute, and stained by the Bodian method to bring out nerve fibers and other details of structure. The Bodian series form the basis of most of the figures in the present report. It would be preferable to have figures and descriptions all based on tested specimens, but comparison of the differentiation of the cochleae of the tested animals with those of the untested series of corresponding ages show so considerable a degree of uniformity at corresponding stages that the use of histological pictures from the untested animals appears to be justified because of the greater detail provided by the special technic employed.

For the sake of brevity and for facility of comparison of the different stages, the description of the cochlea after hearing has been established will be based on the 51-day opossum, and will be followed by an account of corresponding parts in younger and older stages. Wada[29] has given detailed measurements of the parts of the cochlea at various stages of growth in the rat, and Hensen,[11] Böttcher,[4] Retzius,[24] Held,[9] and others have recorded similar measurements in embryos or adults of other mammals. We have not undertaken such measurements in the opossum pouch young, but instead have attempted to fill a gap by noting the degree of differentiation of the supporting apparatus of the organ of Corti and of the

TABLE I.

Showing Tones and Intensity at Different Ages Giving a Standard Response of 1 Microvolt in Tested Opossum Cochleae
Tone in Double Cycles Per Second and Intensity in Decibels

Age of Animal In Days	Cochlea	200 cps	300 cps	500 cps	700 cps	1000 cps	2000 cps	3000 cps	5000 cps	7000 cps	10,000 cps	Highest Tone Eliciting Response
48	Right	.....	.....	.....	.....	47 db	38 db	41 db	41.5 db	45 db	.....	7,000 cps
51	Left	.....	.....	.....	.....	46.5 "	45 "	41 "	41.5 "	.....	.....	5,000 "
	Right	.....	.....	.....	.....	40 "	43 "	36 "	34 "	.....	.....	7,000 "
54	Left	.....	.....	32.5 db	31 db	30 "	27 "	29.5 "	33 "	39 db	.....	7,000 "
	Right	24 db	30 db	30 "	24 "	17 "	24.5 "	38 "	31 "	29 "	35 db	14,000 "
57	Left	23 "	30.5 "	19.5 "	18 "	20.5 "	27 "	16 "	32 "	31 "	33 "	12,000 "
60	Left	17 "	20 "	32 "	26 "	16 "	29 "	10 "	23 "	21 "	32 "	16,000 "
	Right	20 "	21.5 "	17 "	9 "	19 "	14 "	18 "	25 "	19 "	19 "	16,000 "
63	Left	.....	24 "	25 "	17 "	−2 "	−0.5 "	5 "	5 "	1 "	3 "	16,000 "
	Right	.....	17.5 "	16.5 "	14 "	8 "	1 "	−1 "	3 "	−8 "	−1 "	16,000 "
70	Left	.....	29 "	24 "	22 "	18 "	6 "	13 "	20.5 "	9 "	9 "	14,000 "
	Right	17 "	12 "	12 "	12 "	11 "	−3.5 "	9 "	14 "	15 "	23.5 "	12,000 "
77	Left	.....	21.5 "	17 "	13 "	8 "	−5 "	−10 "	−20 "	−23 "	−20 "	19,000 "
	Right	.....	14 "	12 "	10 "	3 "	−8 "	−8 "	−22 "	−26 "	−23 "	26,000 "

tunnel in the several turns of the cochlea at various stages and to correlate these observations with the range of hearing, as measured by oscillographic methods. Attention has also been directed to the development of the hair cells but the technic employed was not favorable for study of their finer structure which might give some clue as to when they become functional. The tectorial membrane in various animals has been studied intensively by Hardesty[7,8], Shambaugh [25] and others. In our preparations, it is so shrunken and distorted by the method of fixation employed that only its extent in the cochlear coils will be indicated, but it is attached to the spiral organ in many of the sections. The basilar membrane does not show any obvious changes in the stages studied, save increase in length and breadth with the growth of the cochlea.

Fifty-one day opossum. The left tested cochlea shows some differentiation of the organ of Corti at the beginning of the apical coil. The tunnel is recognizable as a narrow cleft to a level about 25 sections 16 microns thick, i.e., 0.4 mm., from the apical tip of the cochlear duct. The middle coil and the upper part of the basal coil show differentiation of the organ of Corti, but the tunnel is poorly shown below the middle of the basal coil. The lower extremity of the cochlear duct rests on

FIG. 1 — Photomicrograph of vertical section of cochlea of 51 day pouch young opossum. Bodian stain.

bone, the scala tympani continuing as a narrow caecum beneath a bony ridge. The right tested cochlea shows the organ of Corti differentiated as far as the beginning of the apical coil with undifferentiated epithelium beyond this level to the tip of the cochlear duct.

In Bodian sections of the cochlea of the 51-day opossum (fig. 1) the organ of Corti and the tectorial membrane are present in all the turns, save the distal part of the apical coil. Here the epithelium is of low columnar type, without differentiation (fig. 2A). This picture is characteristic of the distal third of the apical coil at 51 days. At about the middle third of the apical coil, at this stage, the epithelium becomes taller and heaps up at the position of the basilar membrane later to be occupied by the tunnel and its surrounding cells. A few sections below this point, reading toward the middle coil, a slit appears with tall cells on each side (fig. 4AA). They have a differentiation somewhat more advanced than that in the proximal zone of the apical coil in the 48-day opossum (fig. 8B). The slit is the tunnel of Corti and the tall epithelial cells represent an early stage in the differentiation of the pillars. It will be noted that the bases of the pillars are broad and that the distal ends of the cells taper and are elongated with a slight lateral bend. They end against

FIG. 2 — Photomicrographs of sections of cochlear duct and organ of Corti in 51 day opossum. Bodian stain.
A — Distal third of apical coil.
B — Proximal part of apical coil (right) and distal part of middle coil (left.)

the cuticular membrane, which is present as a thin birefringent layer over the entire organ of Corti in early stages of its development. In the more fully differentiated portions of the spiral organ in later stages, the head processes appear to fuse with the cuticular membrane, and, together with the phalangeal processes of the cells of Deiters, form the reticular plate (fig. 4G). A few sections farther on, in the 51-day pouch young (fig. 4A) the bases of the pillers are greatly widened, and the phalangeal cells show a slight tapering toward the cuticular membrane. The hair cells are more differentiated. In the proximal part of the apical coil (fig. 2B, right) the slit has become a narrow triangular space and the hair cells are still further differentiated. The phalangeal cells taper more sharply.

FIG. 3 — Photomicrographs of sections of middle and basal coils in 51 day opossum. Bodian stain.
A — Middle coil.
B — Distal part of basal coil.
C — Middle part of basal coil.

A short distance proximal to this level, i.e., toward the base of the cochlea, the organ of Corti shows a widening of the tunnel, which here assumes a roughly quadrilateral outline, but with rounded corners (figs. 4A and 10). The bases of the pillars are much broadened on the basilar membrane and the distal parts of these cells are enlarged and begin to show convexity in the outer pillars and concavity in the inner pillars. Some of the phalangeal cells show a division (fig. 4A) under the cuticular membrane, the beginning of the phalanges of the reticular plate. Still farther, reading toward the base, the middle coil (figs. 4B and 10) has a relatively large tunnel of Corti, roughly triangular in transverse sections. The pillars are tall and their heads are more differentiated, that of the inner pillar being elongated into a thin, concave process under the cuticular membrane, while the outer pillar shows a terminal enlargement with convex upper surface which fits into the concavity of the opposite pillar in the manner of an articulation. The pillars show the supporting hillocks and tonofibrils, which are beginning to appear at this level, and the heads show dark masses, the ellipsoid bodies, which apparently represent differentiating tonofibrils. Some of the phalangeal cells show the beginning of protoplasmic shoulders for support of the hair cells. In the distal part of the basal coil and the adjacent region of the middle coil (figs. 3A, 4C and 10) the pillars and phalangeal cells approach the adult degree of differentia-

tion. Farther downward, in the basal coil (figs. 4D, 4E and 10) the tunnel gradually becomes smaller and more rounded, and the pillars and supporting cells show less differentiation, with absence of the dark staining ellipsoid bodies, and of the supporting hillocks toward the proximal part of the basal coil. At about the middle of this coil the pillars begin to lose the marked concavo-convex pattern of their distal ends and assume a juvenile appearance similar to that in the proximal apical coil. This is increasingly marked toward the proximal part of the basal coil (fig. 4E) and the tunnel becomes a somewhat rounded space. The hair cells gradually are reduced in size. Nerve fibers are present in all the coils, but in the apical coil they do not extend beyond the zone of differentiation of hair cells.

Forty-eight day pouch young. The middle part of the apical coil shows an early stage of differentiation of the organ of Corti. The tunnel is a mere slit

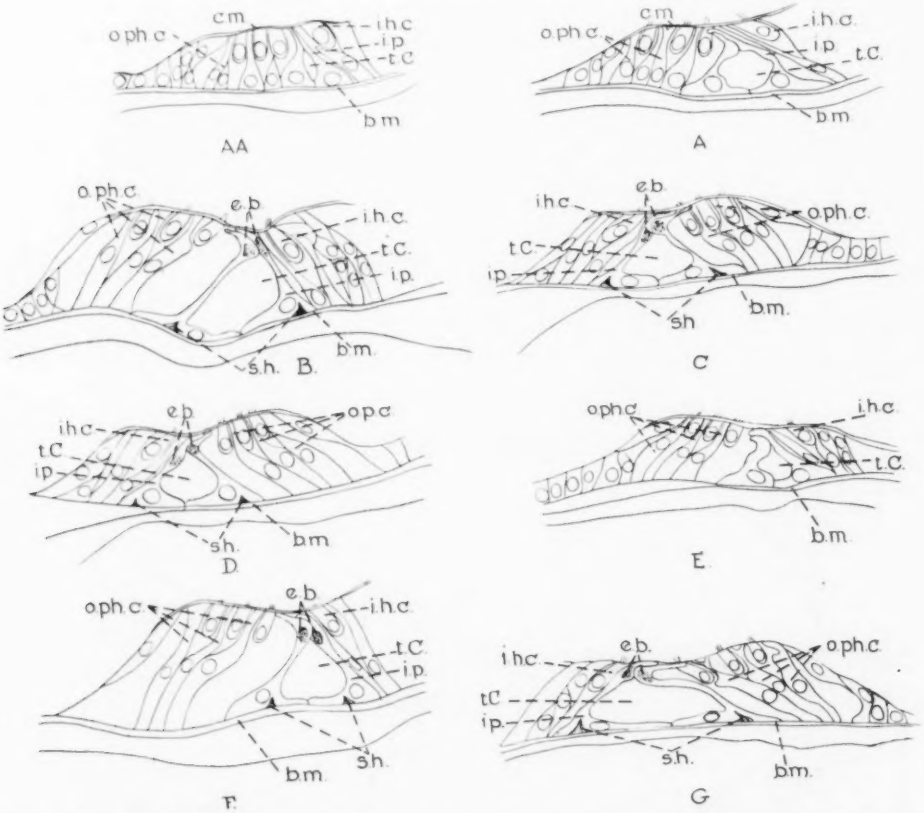

FIG. 4 — Organ of Corti of 51, 48 and 60 day opossums, showing tunnel and pillars in greater detail, Bodian stain. Camera lucida, × 320.

AA — Apical coil, farthest point reached by nerve fibers at 51 days.
A — Transition zone from middle to apical coils at 51 days, ca. level 4A of figure 10.
B — Distal middle coil at 51 days, ca. level 4B of figure 10.
C — Distal part of basal coil at 51 days, ca. level 4C of figure 10.
D — Upper part of middle third of basal coil at 51 days, ca. level 4D of figure 10.
E — Lower third of basal coil at 51 days, ca. level 4E of figure 10.
F — Middle third of middle coil at 48 days.
G — Lower part of apical coil at 60 days.

(figs. 8B and 10) and even this disappears apically. The tectorial membrane extends only to the first part of the apical coil. In the adult opossum this coil has but a quarter of a turn, and somewhat less at 48 days. Toward the middle of the second coil the tunnel broadens and the pillars show early stages of differentiation. Reading toward the basal coil, the pillars appear more and more mature (figs. 8C, 8D, 8E and 10). In the proximal part of the middle coil and the distal basal coil the tunnel appears quite well differentiated (figs. 8E and 10) and the pillars show the tonofibrillar structures, which, however, are absent above and below this zone. Below this level there is a gradual narrowing of the tunnel. This feature is increasingly apparent as the base of the cochlea is approached, due in part to the reduced width of the basilar membrane but largely to an early stage of differentiation. In the middle zone of the basal coil the tunnel and pillars assume a juvenile appearance (fig. 8F) and the tunnel becomes smaller and more slit-like the nearer the cochlear base is approached (figs. 8G, 8H and 10). Comparison of the pillars, as shown in figures 8C (48 days) and 4B (51 days), from approximately the same level of the cochlea,

FIG. 5 — Photomicrographs of sections of apical coil in 54 day opossum. Bodian stain.

A — Distal part of apical coil, near the tip, showing undifferentiated epithelium.

B — Right, distal part of apical coil, proximal to A, showing early differentiation of epithelium of organ of Corti and a narrow slit representing the tunnel at this level. Left, distal part of middle coil.

shows that these structures in the middle coil at 48 days are much less developed, corresponding more nearly to the tunnel and pillars in the transition to the apical coil of the 51-day opossum (fig. 4A), save that the pillars are more differentiated in the latter.

The middle third of the middle coil at 48 days (figs. 8D and 10) shows a differentiation of pillars corresponding approximately to the distal third of the middle coil at 51 days (figs. 4B and 10) but the tunnel is much smaller in the 48-day pouch young and the heads of the pillars less differentiated. The region of transition of the middle coil into the basal coil is well differentiated, both as to pillars and tunnel, resembling the condition in the corresponding level at 51 days (cf. figs. 4C, 8E and 10), but the heads of the pillars appear less developed in the younger opossum. The hair cells are well differentiated in the upper third of the basal coil and in the middle coil, but are small and juvenile in the first part of the apical coil and below the middle zone of the basal coil.

Fifty-four day opossum. The middle zone of the apical coil (figs. 9A and 10) shows a fairly wide triangular tunnel. The bases of the pillars have the characteristic feet on the basilar membrane, but the distal ends show only the first intimation of curving and differentiation. The distal part of the middle coil (figs. 9B and 10) has a broad tunnel and an articulation-like arrangement of the pillars. Early differentiation of the organ of Corti extends to within about 0.2 mm. of the apical end of the cochlear duct (fig. 5B, right) where a narrow slit apparently represents the first indication of the tunnel. No differentiated pillars nor hair cells, and no tectorial membrane are present in this region. The remaining distal part of the apical coil shows no differentiation of specialized epithelium (fig. 5A).

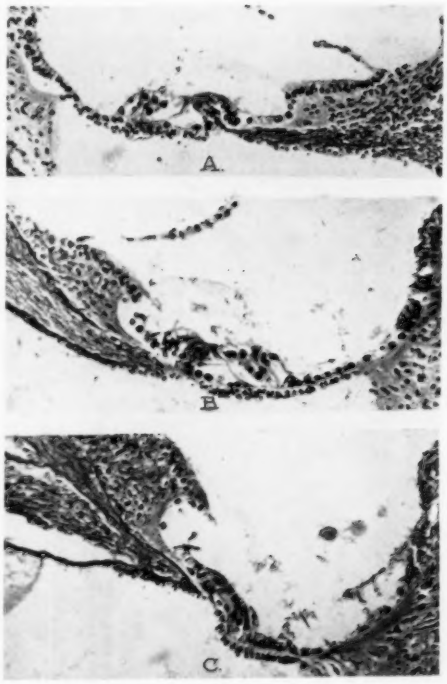

FIG. 6 — Photomicrographs of sections of middle and basal coils in 54 day opossum. Bodian stain.
A — Middle coil.
B — Distal part of basal coil.
C — Proximal part of basal coil.

The middle coil at this stage is well differentiated throughout (figs. 5B, left, and 6A) and the distal third of the basal coil has a well developed organ of Corti (fig. 6B). The middle zone of this coil shows more differentiation than the earlier stages, as does also its proximal part, but there is less differentiation of pillars and phalangeal cells and a smaller and more slit-like tunnel the nearer the base of the cochlea is approached (figs. 6C and 9D). Hair cells are well developed throughout, as shown in figure 6, save in the distal apical and the most proximal part of the basal coils.

Fifty-seven day opossum. At this stage the proximal part of the apical coil is well differentiated, and the organ of Corti, apparently functional, extends to within 128 microns from the apical

end of the cochlear duct. The middle coil is well differentiated throughout, and in the basal coil good differentiation extends to about the middle portion, with progressively less development below this level, so far as could be judged from torn sections. In the right cochlea, of the same animal, not tested, apparently functional organ of Corti extends to the lower part of the middle third of the basal coil.

Sixty-day opossum. The apical coils in both cochleae show differentiation of the organ of Corti proximally, with a low tunnel, broad at its base. The distal part of this coil shows a heaping up of epithelium, but only a slit-like tunnel. The last section, apically, in which the organ can be recognized is 160 microns from the tip of the cochlear duct. The tunnel and the tectorial membrane appear in the next section, downward, and

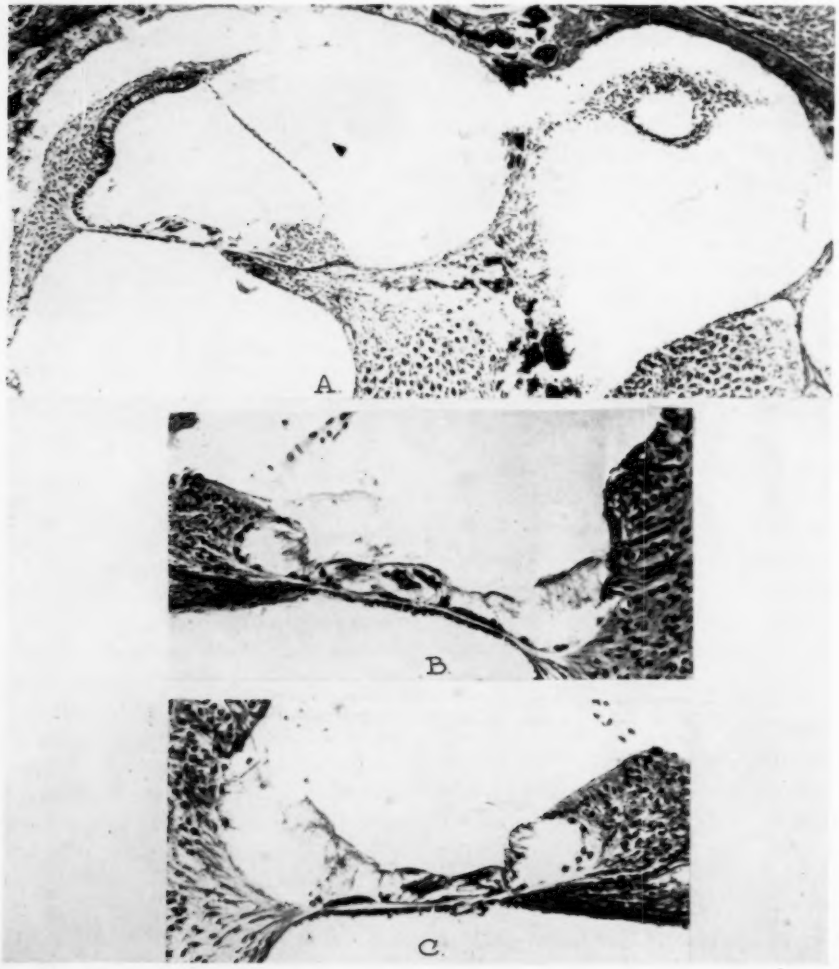

FIG. 7 — Photomicrographs of cochlea of 60 day opossum. Bodian stain.
A — Tip of apical coil (right) and distal part of middle coil (left)
B — Middle part of basal coil.
C — Proximal part of basal coil.

a few sections farther toward the base well defined pillars and a broadening of the tunnel are apparent.

The tunnel and pillars are well differentiated in the distal basal and the middle coils (fig. 7A, left) in which region the organ of Corti is most mature. At about the middle of the basal coil, the degree of differentiation of pillars and tunnel is shown in figure 9E. The tunnel continues as a relatively broad space well toward the oval window, but the pillars are more juvenile in appearance in the proximal zone (fig. 9F). The organ of Corti shows only early stages of differentiation in the proximal third of the basal coil. The tunnel becomes smaller toward the base, as seen by comparison of figures 9F and 7C. The hair cells cannot be distinguished from other cells in the lower part of the basal coil, but the pillars, while small, are distinguishable. However, they are not differentiated into the articulation characteristic of their distal ends in the more mature regions of the cochlea. The most proximal part of the coil rests on bone and the scala tympani ends as a small diverticulum beneath a bony shelf.

Sixty-three day opossum. The organ of Corti is well differentiated in the proximal part of the apical coil on the right side, but how far toward the apex could not be determined because of torn sections. Also the distal and middle parts of the basal coil show good differentiation, but the proximal part is juvenile, with a narrow tunnel. Hair cells and pillars can be distinguished in Bodian sections, but the pillars are small and the hair cells are not greatly different from the neighboring supporting cells. The middle coil shows well advanced development of all its elements. The tunnel reaches the basal end of the cochlear duct as a triangular space (figs. 9G and 10) but of small size due to narrowing of the basilar membrane. The distal ends of the pillars in this region show the beginning of differentiation, as illustrated in the figure. The hair cells are small. The tonofibrillar structures are present both in the apical and the basal coils.

Seventy-day opossum. At 77 days growth appears to be complete. The pillars are tall, save at the tip of the apical coil, and the tunnel is differentiated in all the coils, but has not yet extended to the tip of the apical coil. This may account for the fact that the lowest notes registered in the opossum tests were about 200 c.p.s., although Stevens, Davis and Lurie[26] and Culler[5] obtained responses in the guinea-pig as low as 62 c.p.s. The shorter apical coil of the opossum may of course account for the limitation of range in the low tones.

DISCUSSION

A review of the details of the evidence now at hand concerning the functions of the various elements of the auditory mechanism is not necessary since this subject, together with the more recent developments relating to theories of hearing have been excellently treated by Stevens and Davis.[27] These authors conclude in favor of localization of stimulation in the organ of Corti. Walzl and Bordley[30] have demonstrated the arrangement of successive octaves at equal intervals in the cochlea of the cat. Woolsey and Walzl[35] in the cat, and Tunturi[28] in the dog have shown a point to point projection to the auditory cerebral cortex. Tunturi's studies have shown, furthermore, that pure tones of given frequencies produce activity in the same zones of the dog's auditory cortex as does electrical stimulation applied to the appropriate part of the chochlea.

It is now so well established in several animals as well as in man that the basal coil mediates the higher tones, the middle coil those of intermediate range, and the apical coil the lower tones of the scale, that we may assume that this arrangement holds true in the opossum

cochlea also. In the 48 and 51 day pouch young, zone 1 (fig. 11) had attained the highest degree of differentiation. At 48 days apparently functional differentiation extended apically to zone 2, and at 51 days to zone 3. In the basal coil functional differentiation at 51 days extended to zone 7 of figure 11. The limits of response at 48 days, as shown in table I, were 1000 c.p.s. and 7000 c.p.s. at 47 db. At 51 days they were 1000 c.p.s. and 5000 c.p.s. at 46.5 db. The first response noted in the opossum, as already stated, was to about 1300 c.p.s. at 50 days. If the region of greatest differentiation at 48 and 51 days, namely zone 1 of figure 11, may be regarded as serving tones of about this range, we have a starting point for localization of other tones, those of lower pitch apically and higher tones toward the base of the cochlea. The region of reception for 1000 c.p.s. at 48 days may then be regarded as localized in zone 2 (fig. 11), and to have reached zone 3 at 51 days. This must mean either an apical extension of the region of receptivity for this tone or an apical migration of the locus of receptivity.

By 54 days, zone 4 of figure 11 represents the apical limit of differentiation of the supporting apparatus to apparently functional stage. As shown in table I the lower limit of response at this stage was 200 c.p.s. in the right cochlea and 500 c.p.s. in the left, at 24 and 32.5 db., respectively. At 60 days the apical limit of differentiation had reached zone 5, with response in both cochleae to 200 c.p.s., at 17 db. on the left side and at 20 db. on the right. By 63 days differentiation had reached zone 6, with response to 300 c.p.s., at 24 db. and at 17.5 db., on the left and right sides, respectively. Zone 6 is somewhat proximal to the region of the apical coil indicated by Stevens and Davis[27] (figure 140), as the locus of 250 c.p.s. in man. Possibly further differentiation in the pouch young would advance it more apically, but our sections of stages subsequent to 60 days were not favorable for accurate determination of this point. The stages described indicate that along with apical differentiation of the supporting apparatus there is either an extension or a migration apically of the 200 to 300 c.p.s. range, comparable to that of the 1000 c.p.s. range in earlier stages. The evidence for localization of tonal ranges in adult animals (Culler,[5] Stevens and Davis,[27] p. 370) favors the interpretation that migration rather than extension occurs. Such a migration of locus of receptivity appears most readily explainable, on the theory of tonal localization in the organ of Corti, by increased elaboration of the supporting structure in the apical direction.

Our sections of the greater part of the basal coil in the 48 day pouch young were not favorable for determining the lower limit of differentiation of the spiral organ, but an apparently functional stage of differentiation of the pillars and tunnel had reached zone 7 of figure 11 at 51 days. The highest limit of response at this stage was 5000 c.p.s. at 34 db., although at 48 days it was 7000 c.p.s. at 45 db. In the 54 day pouch young the high limits were 7000 c.p.s. and 14,000 c.p.s. for the left and right ears, at 39 and 29 d.b., respectively, and differentiation had reached zone 8 in the left cochlea. The 60 day pouch young responded to 16,000 c.p.s. at 31.5 db. and differentiation of pillars and tunnel had reached zone 9. In subsequent stages, as described above, differentiation continued toward the base, and the 77 day pouch young responded to a maximum of 26,000 c.p.s. Whether or not there was any migration of locus of response in the basal coil, with growth of the cochlea, corresonding to that described in the middle and apical coils we have no evidence. Assuming, as our observations and those of Wada[29] and Lane[14] seem

to indicate, that a certain degree of differentiation of the tunnel and pillars of Corti and, we would add, the remainder of the supporting apparatus, is necessary for functioning of the auditory mechanism in mammals, the zone of disturbance of the membrane necessary to a given tone appears to migrate with development of the organ. If the basilar membrane determines localization this might be accounted for on the ground of increased breadth of the membrane with growth. However, Wever[31] has shown that in the apical coil the basilar membrane tapers rapidly toward its terminus. Another possible explanation is that the supporting apparatus plays some role in conjunction with the basilar membrane and other structures, a role which becomes effective only as these elements attain the necessary degree of differentiation.

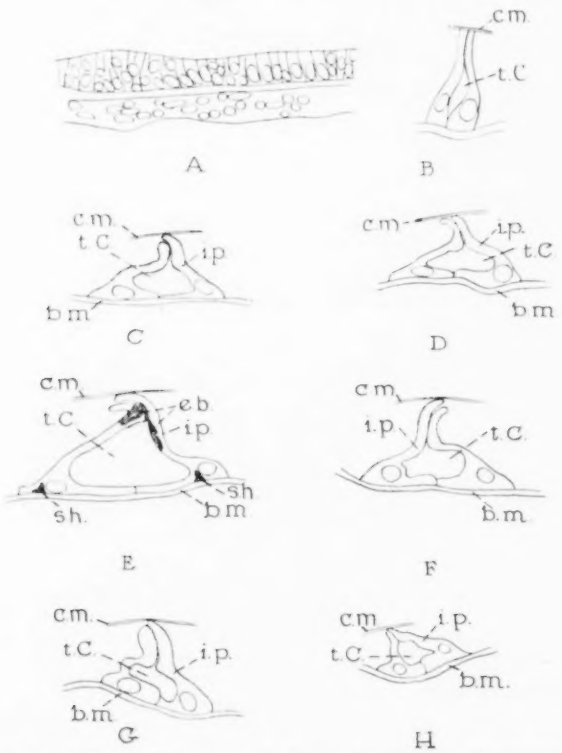

FIG. 8 — Stages of development of tunnel and pillars of Corti at various levels of cochlea in 48 day opossum. Camera lucida, × 386.

A — Undifferentiated epithelium in distal half of apical coil, ca. level 8A of figure 10.

B — Early differentiation of tunnel and pillars in middle of apical coil, ca. level 8B of figure 10.

C — Tunnel and pillars in distal portion of middle coil, ca. level 8C of figure 10.

D — Tunnel and pillars in middle third of middle coil, ca. level 8D of figure 10.

E — Tunnel and pillars in transition zone from basal to middle coils, ca. level 8E of figure 10.

F — Tunnel and pillars in upper third of basal coil, ca. level 8F of figure 10.

G — Tunnel and pillars about middle of basal coil, ca. level 8G of figure 10.

H — Tunnel and pillars near base of basal coil, ca. level 8H of figure 10.

Concerning the function of the pillars of Corti and, with them, of the tunnel, various opinions have been expressed. The fact that birds have no pillars, although many of them produce and no doubt hear a considerable range of notes, is the great stumbling block in the way of ascribing a function to these structures. Helmholtz, in his first enunciation of the resonance theory of hearing, postulated that the pillars are the resonators. He subsequently changed this view in favor of the fibers of the basilar membrane. Ewald[6] held that the pillars constitute a tension apparatus for the zona arcuata of the basilar membrane, this zone forming the floor of the tunnel. He considered the pillars as contractile elements whose movements regulate the size of the tunnel. Held,[10] on

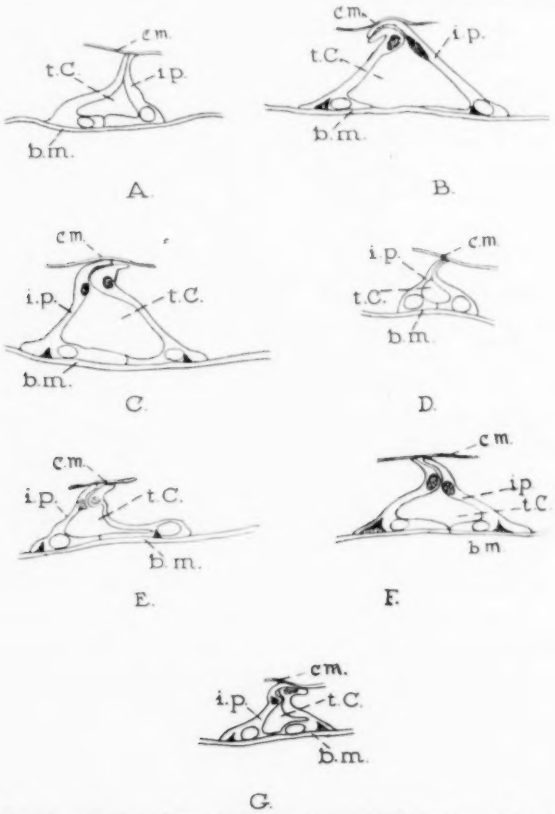

FIG. 9 — Stages of development of tunnel and pillars of Corti in 54, 60 and 63 day opossum. Camera lucida, × 386.

A — At about the middle of the apical coil, ca. level 9A of figure 10, in 54 day opossum.
B — Distal part of middle coil, ca. level 9B of figure 10, in 54 day opossum.
C — Proximal part of middle coil, ca. level 9C of figure 10, in 54 day opossum.
D — Proximal part of basal coil, ca. level 9D of figure 10, at 54 days.
E — At about middle of basal coil, ca. level 9E of figure 10, in 60 day opossum.
F — Proximal part of basal coil, ca. level 9F of figure 10, in 60 day opossum.
G — Proximal part of basal coil, ca. level 9G of figure 10, at 63 days.

the basis of histological studies of the organ of Corti, was unable to accept Ewald's conception of the zona arcuata as the vibrating portion of the basilar membrane or of the tunnel as a regulating accommodation apparatus. Kolmer[13] describes the pillars in considerable detail but states that the articulation-like arrangement of their heads is not movable. Our preparations show that the heads of the pillars are not fused together (figs. 8 and 9), but have the appearance of an articulation. Their flattened processes form the inner part of the reticular plate, to which the pillars give the principal and more rigid support.

The outer border of the reticular plate is attached to the inward bending row of outer phalangeal cells which have long curving tonofibrils whose ends spread out in the border of the reticular plate. The outer supporting structure thus appears much less rigid, the tonofibrils of the outermost row of phalangeal cells suggesting a spring-like apparatus. The phalangeal cells, with their curved bundles of tonofibrils extending to the reticular plate and spreading out in the phalanges, support the reticular plate between the two borders as a series of minute springs. Based on this conception, the reticular plate is so supported that its inner border appears relatively immobile, save as the pillars may bend slightly, while toward its outer border it seems capable of greater movement. This would result in gradations of elasticity in the reticular plate.

Retzius,[24] Shambaugh,[25] Prentiss,[21] Kishi[12] and others, from histological studies, held that the tectorial membrane is attached to the spiral organ, while Shambaugh, Prentiss and others, state that the hairs of the receptor cells also attach to the tectorial membrane. It therefore appears reasonable to assume, if these views are correct, that the pillars, the reticular plate and the phalan-

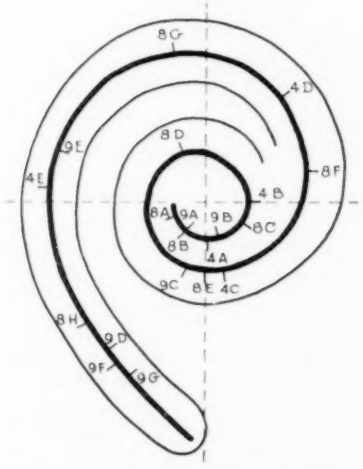

FIG. 10 — Diagram of cochlea showing approximate levels of sections of the organ of Corti or of tunnel and pillars illustrated in figures 4, 8 and 9. The various levels are numbered to correspond with the numbers of the figures.

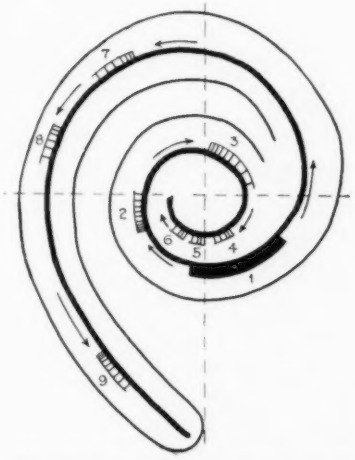

FIG. 11 — Diagram of the cochlea to show the zones of apparently functional differentiation reached at various stages of development, as described in the text. Zone 1 represents the region of greatest differentiation of the organ of Corti, confined to the distal basal coil and the neighboring part of the middle coil, in the 48 day opossum. The arrows indicate the direction of subsequent differentiation.

geal cells may function as a cushioning and tension apparatus between the tectorial membrane and the basilar membrane to maintain a normal optimum tension of the cilia. If the tectorial membrane is attached along Hensen's stripe and at its outer border, as many observations indicate, the main part of it thus rests on the reticular plate against the projecting ends of the hair cells. The entire mechanism has the appearance, as illustrated in figure 12, of a supporting apparatus for the tectorial membrane, the reticular plate apparently forming an elastic surface superimposed upon the basilar membrane and presumably modifying and refining such elasticity as the latter may possess.

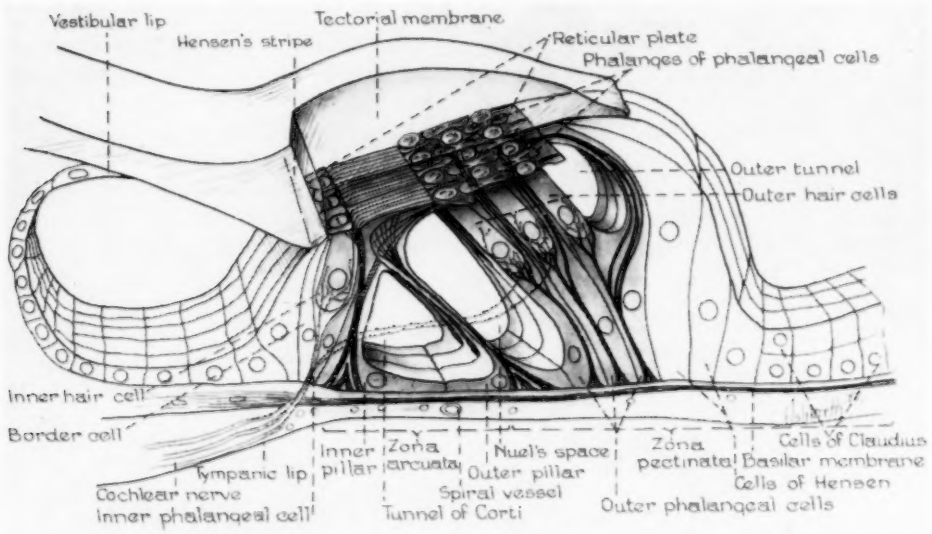

FIG. 12 — Semidiagrammatic representation of the organ of Corti and the tectorial membrane, based on the interpretation that the latter is attached along Hensen's stripe and at its outer margin. The supporting apparatus, reticular plate and hair cells are shaded. A portion of the tectorial membrane is represented as cut away to show more clearly the reticular plate, the phalanges and the ends of the hair cells. The remainder is depicted as translucent. The pillars of Corti and the outer phalangeal cells show the arrangement of the tonofibrils (black lines) and their manner of spreading in the head processes of the pillars, in the phalanges and and below the basal ends of the hair cells. Adapted from Retzius, Held, Testut, Shambaugh and others.

Both basilar membrane and tectorial membrane are present in all vertebrates beginning with the anurans, that is in all types receiving sound waves from the air, but only mammals have the organ of Corti. Both membranes therefore must have basic functions in connection with sound reception. The tonal ranges of amphibians have not been measured. Wever and Bray[32] observed a range of 120 to about 1000 c.p.s. in the turtle (Chrysemys picta). Among birds the pigeon has a range of 100 to 10,000 c.p.s., but with poor efficiency of reception as compared with mammals (Wever and Bray[33]). The increase both in range and sensitivity shown by the opossum pouch young, as the supporting apparatus becomes increasingly differentiated, appears to indicate that this apparatus has a role beyond mere support of the hair cells. The function suggested, namely, increasing the elasticity of the cochlear wall through resilient support of the reticular plate, together with the concept that the tectorial membrane rests upon the reticular plate and is attached to the inner border cells and the

cells of Hensen, gives meaning to the entire ensemble of the organ of Corti and its related structures, which must function as a unit.

Under this conception the pillars and the phalangeal cells would maintain a constant optimum pressure between the tectorial and basilar membranes, momentarily modifiable by movements of segments of the basilar membrane. If this be true, stimulation of the hair-cells could be brought about by increased pressure, beyond the normal, or by pulling of the hairs attached to the tectorial membrane, as the basilar membrane vibrates downward. A continuously intimate association of the tectorial membrane with the rest of the receptor mechanism would appear necessary to account for the extreme sensitivity of the organ of Corti to minute vibrations.

Comparison of the sound intensities required at different stages of development of the opossum (table I) with the stages of development of the pillars, the tunnel and the supporting apparatus in the various coils (figs. 4, 8 and 9) appears to indicate that continued differentiation of this apparatus serves to increase the sensitivity of the organ of Corti. In the earlier stages of development, when the tunnel and supporting apparatus are poorly developed, the sound intensity required to produce a response of 1 microvolt, as shown by McCrady, Wever and Bray,[20] was many times greater than in later stages of the opossum pouch young. Also the tonal ranges requiring the lesser intensities of sound at different ages of the pouch young lie in the regions assigned to these ranges on the basis of order of development, while the ranges both above and below these tones required a greater intensity. Likewise it will be noted from table I that up to 70 days the intensity required at all the tones tested, in general, decreases with age and therefore with differentiation of the organ of Corti. The 70 day pouch young is a partial exception to the trend, but appears to be a case of slower individual development, as suggested by McCrady, Wever and Bray. In the 77 day opossum, while the 2000 c.p.s. tone required the least intensity (–5 and –8 db.) of any of the stages studied, the peak receptivity had shifted to 7000 c.p.s. at –23 and –26 db. for the left and right ears, respectively. McCrady, Wever and Bray reported a similar increase in sensitivity in older stages (78 to 82 days) of the Mexican opossum at 7000 c.p.s. range, and in their later report, state that this zone is the point of maximum sensitivity for the adult opossum also. The maturing of the spiral organ in the lower part of the basal coil presents the most obvious explanation.

The interpretation that the organ of Corti increases sensitivity is strengthened by the fact that in the pigeon which, like other birds, has no pillars or tunnel, Wever and Bray found a markedly low efficiency of the cochlea as a transmitter of sound. This is true in spite of the fact that birds have many more rows of hair cells at a given level of the papilla basilaris than do mammals, as shown by Held.[9] The organ of Corti must therefore largely be responsible for the greater efficiency of the mammalian cochlea, although the differences between the ossicles of the mammalian middle ear and the columella of birds must not be omitted from consideration.

Wilska,[34] has recorded vibration amplitudes of the human tympanic membrane, producing audible tones, ranging from 1830 millimicrons at 45 c.p.s. to as small as 0.0045 millimicrons at 3600 c.p.s. Lord Rayleigh[22] many years ago had pointed out that only minute amplitudes were necessary. With Wilska's results as a basis, Stevens and Davis[27] (p. 56) have computed the displacement of the basilar membrane, at near 3000 cycles, as less than 1 per cent

of the diameter of a hydrogen molecule. It would be difficult to conceive of so infinitesimal a displacement being transmitted through the organ of Corti to the hair cells so as to cause them to impinge against, or pull from, the tectorial membrane with sufficient force to produce a piezo-electric effect, even if the whole system had the incompressibility of steel. Since this must be far from the case in the organ of Corti by reason of the structural arrangement of the supporting apparatus above described, and also in the tectorial membrane because of its structure, one may be allowed to question the vibration factor of the basilar membrane as the sole cause of hair cell stimulation. This does not deny movement of the basilar membrane in response to vibration in the perilymph, but merely raises the question as to the primacy of such movement as the stimulus producing factor.

Békésy,[2,3] made a simplified model of the ear in the form of a brass box, with a medial partition also of brass. Part of the partition, however, was made of an elastic membrane, representing the basilar membrane. This investigator then determined, by stroboscopic illumination, the form of oscillation of the membrane to sounds of various frequency. Standing waves were produced with several nodes, the internodal intervals being of different lengths and increasing from the base toward the apex. Békésy subsequently studied the human ear by observing the oscillations of the basilar membrane with a microscope through a hole in the bony cochlea, using stroboscopic illumination. He extended the validity of his earlier experimental results to the human cochlea.

Reboul[23] computed by mathematicophysical formulae the propagation of concussions in a fluid column, applying the laws of the mechanics of fluids. For the sake of as great simplicity as possible he assumed that the scala vestibuli is a conical tube in which the fluid (perilymph) is periodically compressed and distended by oscillatory movements of the membrane at the oval window. If the vibrations of compression are so rapid that they cannot be transmitted through the narrow helicotrema to the scala tympani, the oscillations are confined to the scala vestibuli, which is limited above by a solid wall, the bony cochlea, and below by an elastic wall, the basilar membrane.

Basing his equations on the propagation of the vibratory disturbance in a fluid column contained in a tube with elastic walls, Reboul showed theoretically that for high tones the basilar membrane undergoes maximal displacement at a point which is a function of the frequency of vibration.

Comparing his results with the experimental observations of Békésy, Reboul states that at the lower frequencies, toward the apex, the basilar membrane is displaced "like a vibrating reed," while at higher frequencies near the base, on the contrary, movements of maximal amplitude are located at specific points on the membrane. Reboul's curves based, as he states, upon the smallest possible number of hypotheses, provide a possible explanation for the fact that the ear behaves as if it contained numerous small resonators, each tuned to a specific frequency, but suggests that the real explanation may lie in the fact that the oscillating pressure does not reach the same amplitude at all points along the cochlear duct, but attains maxima at points determined by the frequency of the stimulating sound and the elasticity of its wall.

The evidence for tonal analysis in the cochlea has become so formidable, especially in view of the results of Ades, Mettler and Culler,[1] that the telephone theory insofar as it expresses absence of cochlear analysis, may be omitted from consideration in discussing our results on the opossum. These results, however,

should be viewed against the background of the various conceptions of the cochlea as the analyzer. It must be stated at the outset that most previous discussions have almost entirely omitted from consideration the pillars of Corti and their related structures in the cochlea, save as support for the hair cells, giving chief attention to the basilar membrane and the hair cells.

Our investigations show that appearance of function of the organ of Corti, as indicated both by reflex tests and by the oscillograph, is closely associated with the development of the supporting apparatus and the tunnel of Corti. If we may regard this apparatus, as previously suggested, as a cushioning and tension mechanism, supplementing and refining the elasticity of the basilar membrane upon which it rests, some of the difficulties in arriving at a rational explanation of the inner ear may be removed. Since histologists have described the pillars of Corti as so interlocked as to make individual movement impossible, vibrations of specific positions of the basilar membrane, as postulated by the resonance theory, cannot be transmitted to specific hair cells. In the opossum pouch young the basilar membrane is developed throughout the length of the cochlea quite early. It does not appear to be any different in sections of the nonfunctional 29-day opossum and those of 48 days and subsequently, after function has been established, although special methods of technic might show differences in its fibers. However, so far as we can judge from the material at hand, if it vibrates at all, there is no reason why it should not vibrate much earlier than 48 days. There are, in contrast, striking changes in the organ of Corti as described in the earlier part of this paper.

If these changes may be interpreted as completing the differentiation of the duct system of the auditory apparatus as elastic tubes in which waves in the perilymph and endolymph can become effective as stimulators when the proper degree of differentiation is brought about, then we can ascribe a function to each of the parts of the complex auditory mechanism, instead of giving the basilar membrane alone the stimulating function. In such a conception pressure in the perilymph of the scala vestibuli, transmitted through Reissner's membrane and the endolymph and localized according to the pressure-gradients determined by the frequency of the soundwave, would impinge on the tectorial membrane and through it, on the hair cells. Assuming that the tectorial membrane is attached to the organ of Corti and that the cilia extend into this membrane, no primary motion of vibration would be necessary by either the basilar or the tectorial membrane to stimulate the receptors, since the stimulus would be a localized concussion. Whether or not this conception is capable of experimental approach, it has the advantage of taking into consideration all of the anatomical factors of the cochlea.

SUMMARY AND CONCLUSIONS

Our investigations may be summarized as follows:

The earliest differentiation of the organ of Corti to apparently functional stage was found in the upper basal and the adjacent lower medial coils. This differentiation consists in an elaboration of the pillars of Corti, the tunnel, the phalangeal cells and the reticular plate to a condition approaching adult structure, and in enlargement of the hair cells. As the opossums continued to develop, differentiation of the organ of Corti extended both apically and basally from this initial zone. Pari passu, there was an increase in tonal range from an initial zone of 1000 to 1300 c.p.s., to a range of 200 to 26,000 c.p.s. A lowering of sound intensity required for response

to notes of given frequency also accompanied development and appears to be correlated with the histogenesis of the elements of the organ of Corti.

The differentiation of the organ of Corti, especially the pillars and supporting apparatus is interpreted as gradually modifying and refining the elasticity of the basilar membrane, thereby making the mammalian cochlea more efficient as a sound receptor, as compared with birds, which do not possess an organ of Corti, and also in some way extending the tonal range. The reticular plate is regarded as a resilient surface upon which the tectorial membrane rests and through which the hair cells project. Momentary modifications of its tension brought about by waves in the basilar membrane, are suggested as a factor in the phenomena of tone analysis.

Because the structural characteristics of the adult cochlea as well as the phenomena of differentiation and of receptivity of the developing organ, as shown in the opossum, leave much to be explained under the conception that the tone analyzing mechanism is composed of a series of individual resonators in the basilar membrane, some other physical principle, possibly similar to the theoretical waves of Reboul, should be considered as offering a more satisfactory explanation of tonal localization.

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DISCUSSION

ARNOLD A. ZIMMERMAN, Sc.D., Chicago: A main fact is at once apparent from the study by Drs. Larsell and McCrady, namely, that the labyrinth of marsupials, of which the opossum is a primitive representative, has attained a high degree of histologic and functional differentiation. Until recent years little had been known concerning the histologic details of the cochlea in these animals. The labyrinth had been merely described as resembling that of Insectivores. The main reason for using the opossum in anatomic research lies of course in the fact that it offers unusual opportunities to study function during development.

In a previous study Drs. Larsell and McCrady had given the first detailed account of cochlear differentiation in that species. The present report constitutes an expansion of that study into older developmental stages.

The various methods used in the functional tests were interesting indeed. As the results had been previously published, the technic employed need not concern us in this discussion. The present study deals primarily with a correlation of observed detail in cochleae of developmental stages which corresponded to those functionally tested.

Any critical remarks concerning interpretations of histological features of the organ of Corti must necessarily take into consideration the great technical difficulties which are encountered in such studies. Fixation, decalcification of the petrous bone as well as shrinkage of the soft tissues due to embedding and staining methods are most detrimental processes in any attempts at preserving the delicate structures of the internal ear.

As most figures shown in this report were based on material treated according to the *Bodian* silver technic of staining, I should like to ask Dr. Larsell whether he has found this method to give superior results as compared to the pyridine-silver technic.

Disturbing distortions are known to be by-products of most silver methods. For this reason it would have been desirable, perhaps, to include in this report some figures obtained from the series of tested cochleae

which were stained with haemotoxylin and eosin.

In the photomicrographs shown good histologic details of the organ of Corti, as a whole, are scarce. To judge from the illustrations of the material used it appears that it would have been particularly difficult to study other components of the complex supporting structures within the basilar papilla. The pillars are perhaps the strongest supporting elements and are more readily retained in their arrangement through various technics of fixation and staining. Yet it should be emphasized that the differentiation to a functional state of other supporting elements probably is concomitant with that of the pillars and in an analysis of tone perception they should perhaps not be altogether omitted.

With regard to the termination of nerve fibers in the functional stages of the cochlear end-organ, the figures obtained by the Bodian technic give us little information not already contained in a previous report on the non-functional cochlea of the pouch-young of 29 days. Nerve endings were shown to be present, in that relatively early stage, about the hair cells of the basal and middle coils when a tunnel of Corti is indicated as a narrow slit. In the report under discussion it is merely stated that "nerve fibers are present in all the coils" of a pouch-young of 51 days, but that "they do not extend beyond the zone of differentiation of the organ of Corti."

This is somewhat disappointing in view of the fact that the Bodian method of staining was used with the definite purpose of "bringing out nerve fibers."

It would have been particularly interesting if in the hair cells of the *functional* cochlea a perinuclear net of a neurofibrillar apparatus could have been demonstrated. Kolmer and others have shown convincingly such a reticulum to exist in hair cells of rats and mice with the aid of the Cajal and Bielshowski silver methods.

In view of the known fact that nerve endings within the organ of Corti are not all radially arranged but may course spirally up and down the basilar papilla it might have been desirable to demonstrate the course of such fibers in the inner and outer spiral strands, by sections cut transversely to the modiolus. Not only is the nerve supply of the supporting elements of the cochlear end-organ obscure, but it is also a debated question whether a single nerve fiber is in relation with one or several portions of the organ of Corti.

Young mice and rats have a simple nerve plexus within the organ of Corti. It might very well be that in opossum pouch-young a still more primitive and simple arrangement of that plexus could be demonstrated in sections of the appropriate plane.

It would seem to me of importance to study the possible spiral course of the afferent fibers of cochlear neurons in view of the shift in locus of tonal perception which Larsell and McCrady have described in this study.

Although nerve endings were present about the bases of hair cells in the basal and middle turns of the cochlea in pouch-young of 29 days, their presence at that degree of differentiation did not indicate function of the particular vibro-receptor (hair cell) concerned. This was proved by acoustic tests on pouch-young of the same developmental stage. The tunnel of Corti was poorly developed and the organ was non-functional. On the other hand, an intracellular net of neurofibrils was not demonstrated by the technics employed for the younger nor the older functional stages. The question presumably is open as to whether non-function of the organ of Corti at that stage *or later* is related to incompletely developed supporting pillars of the tunnel of Corti or whether it might be due to an incompletely differentiated neuro-epithelial end-apparatus.

Possibly the perceptivity is due to the apical extension of dendrites in the outer spiral courses. The further development of the tunnel of Corti and its pillars could occur synchronously but the latter process would not necessarily represent the only or main criterion of functional maturity of the organ of Corti in any one zone.

Comparisons between the degree of differentiation and of function were based primarily on the developmental status of the pillars. On that basis an extension or apical migration of a particular zone of receptivity was postulated.

The explanation for this apparent shift rested exclusively "on the increased elaboration of the pillars and tunnel in the apical direction."

Yet one may ask whether one type of supporting elements of the organ of Corti was not overemphasized with the resulting tendency of explaining tone perception primarily on the basis of physical or mathematico-acoustic phenomena. To be sure, the structural differentiation of the end-organ of hearing must provide conditions by which some such laws may become effective.

In contradistinction to this I would stress, rather, the equally important differentiation of the neuro-epithelial junctions. The excel-

lent Bodian technic might be expected to give further insight into the terminations of afferent cochlear neurons on the hair cells. It is difficult to conceive that relatively well-developed pillars of the tunnel of Corti *but incomplete neuro-epithelial junctions* would lead to tone perception. In this respect the report by Drs. Larsell and McCrady leaves room for further investigations.

That the entire supporting apparatus within the basilar papilla has some degree of elasticity is accepted by most authors. Whether or not the middle segment of the individual pillars would be able "to bend slightly and straighten again by a springlike action of their tonofibrils," as assumed by Larsell and McCrady, might be questioned on the basis of the generally accepted fact that the tonofibrils are densely packed in the middle portion of the pillars, where their arrangement presumably might produce greater strength or stiffness, as compared to the cone-like separation of the fibrils near the basilar membrane and beneath the cuticular headplate.

Drs. Larsell and McCrady express themselves cautiously in stating "that the pillars may function as a cushioning and tension apparatus between the reticular membrane and the basilar membrane." The latter function has been questioned by several authors.

Dr. McCrady and co-authors had also observed previously that the opossum hears tones best of about 7,000 cycles per second. The locus of reception of such relatively high tones presumably should show special characteristics of the pillars, enhancing the efficiency of the organ of Corti. The report under discussion fails to give evidence for such an assumption; nor does it furnish any other explanation for the greater efficiency near the upper end of the tonal range.

The authors made, however, the suggestion that the supporting apparatus of the pillars may serve to increase the *sensitivity* of the organ of Corti in general. In my estimation this is a very different problem from that of the initial tone perception, at *any* intensity of sound, in a particular zone.

Correlations between required tone intensities and development of the pillars appear to give good support to that view.

I would agree, therefore, with the statement by Drs. Larsell and McCrady that "the pillars constitute a supplementary and refining apparatus," particularly if related to *sound intensities* and therefore to the sensitivity of the organ of Corti.

Barry J. Anson, Ph.D., Chicago (by invitation): In the important investigation of which Dr. Larsell's paper forms a part, the authors have employed a unique type of mammal, which becomes an extrauterine "embryo" on the thirteenth day, and almost forty days before it can hear. This means that the opossum is an ideal subject for auditory experimentation; it can be studied while the anatomical mechanism for hearing is undergoing initial development and attaining "adult" perfection.

Dr. Larsell and his colleagues, in this project designed to correlate structure with function, have found that portions of the organ of Corti, namely, pillar cells and tunnel bounded by the latter, are differentiated in the 51 day opossum, at which stage hearing is established; and that receptivity increases with maturation of these histologic elements. They therefore conclude that resiliancy of the pillars permits a movement which, in occurring between the hairs of the receptor cells and the tectorial membrane, maintains an optimum tension in the cilia and increases auditory efficiency.

But, as Dr. Larsell would unhesitatingly admit, other equally delicate elements might be following a comparable course of development. I would like to inquire if, at some future date, we may expect a report from his laboratory upon the concurrent development of tectorial membrane, nerves of the spiral ganglion, the hair cells, nerve fibers in the spiral lamina and in acoustic areas of the cerebrum. One would require such information as a basis for appraisal of function of each portion of the auditory apparatus. Possibly the pillar mechanism is a mere auxiliary.

It may be vain to hope that histologic procedures will ever furnish a "life-like" undistorted picture of this wondrous apparatus. Surely, Dr. Larsell and his associates are in a position of advantage in relation to this research. We trust that they will attempt to close the hiatuses in our knowledge.

Edward McCrady, Jr., Ph.D., Sewanee, Tennessee (by invitation): I want to comment primarily on evidence concerning what makes any ear hear best at one pitch and less well in either direction above or below that pitch. That comes into this particular discussion because we have referred to an increase of sensitization for certain frequencies, for instance, around 7,000 cycles, and we have suggested that this is due to a differentiation of the pillars.

This will, perhaps, be accepted on the evidence given, but there is a further question why a higher sensitivity is eventually reached for 7,000 cycles in the opossum than for other regions which have pillar cells equally well differentiated. In man the same question would apply to the general area, say, between 2,000 and 4,000 cycles.

Only recently I have come across what appears to be the answer to that question. It was partly due to the fact that I have been obliged to teach physics in connection with the war program, and this made me go back to some differential equations which I probably would never have looked at again if I had not been required to. But in the last few years, in connection with radio work, a good deal has been done in the study of propagation of sound waves in different media, and I was able to find evidence that the maximum efficiency of pressure exerted by the energy of a sound wave traveling through a liquid in a tube depends upon the relation of wave length to the circumference of the tube.

That fact has never been associated in any way, has never been considered, I believe, in connection with the problem of hearing, and it occurred to me that it might give us the clue.

To settle that question, I had to calculate the wave lengths of the sounds we hear best, and correlate them with the circumference of the tube in the region where we presume they are received or throughout the cochlea, for that matter.

These calculations turned out to be, at first, discouraging. The figures for transmission of waves in ordinary water were altogether too large. They would mean that the cochlea would have to have a circumference of about 15 feet if my theory were correct. So, I put the notes aside and felt that I had had a misleading brainstorm. But a little later it occurred to me that, as a matter of fact, the rate at which waves travel in a tube (which, of course, determines their wave length) is very different from the rate in open water, and must be calculated from an equation including terms for elasticity of the walls of the tube, thickness of the walls, diameter of the tube, and a few other factors not included in the equation for waves in unconfined media. Values for some of the constants concerned are not easily obtained (e.g. Young's modulus of elasticity for *living* human temporal bone), but by using information from animal experiments I found calculated velocities varying from 10 meters a second to 50 meters a second. But already I saw that this whole range of values was considerably different from 1400 meters a second, the figure I had before.

It turned out finally that, if you took a figure halfway between those extremes and exactly in the middle of the figure set by Davis and Stevens for the velocities of the waves in the basal coil, if, to be specific, you took 28 meters per second and then calculated the diameter of the cochlea which would give maximum efficiency for that velocity and for frequencies of 2,000 to 4,000 cycles it turned out to be precisely the diameter of a human cochlea.

Then I took the extreme dimensions of the cochlea, and these fitted perfectly and showed that, under the conditions existing in man, you would necessarily hear best in that range of pitch because the diameter of the cochlear duct (this was including the scala vestibuli and the cochlear duct proper or scala media) corresponded to those dimentions which gave maximum efficiency in exactly that range. Notes above or below that would be heard less well because the pressure delivered was decreased by the inappropriate dimensions of the tube in which the waves were carried.

This is a new idea which has to be further explored by checking with dimensions in other animals. I did check with dimensions in the opossum. There the new principle predicted what we have discovered, namely, that the maximum efficiency would be in the region of 7,000 cycles.

It may further explain loss of hearing in old age. If in the basal coil there is further extension of diameter of the scala vestibuli with age, this would account for decreasing efficiency of hearing in the regions, say, above 10,000 cycles or, for that matter, regions above 4,000 to 6,000 cycles, which is a familiar fact and, I believe, has heretofore had no sort of explanation.

And finally, it may explain the decreased sensitivity which occurs in Ménière's disease. Dr. Lindsay is exhibiting at this convention photomicrographs showing in such patients a kind of labyrinthine dropsy with increased pressure in the endolymph. My interpretation would be that the increased pressure changes the rate of propagation of sound waves in the cochlea. This in turn changes their wave lengths so that the latter no longer coincide with the circumference of any portion of the tube, and this causes decreased efficiency.

O. LARSELL, Ph.D., Portland, Oregon: With reference to the question Dr. Zimmermann raised, the problem of technic is a very diffi-

cult one, as anyone knows who has worked on the cochlea. We chose the Bodian technic because it showed the cell structure, nuclei, and so on, about as well as hematoxylin-eosin, and it also shows nerve fibers. As compared with the pyridine-silver technic, it does not show the fine fibers that pyridine-silver does, but it shows other structures better.

With reference to the hair cells, we demonstrated them, as Dr. Zimmermann pointed out, in the 29-day opossum several years ago. We could not follow them up in minute detail in the large number of specimens we were investigating.

The study of other supporting elements we did not undertake. Those studies, with careful measurements have been made by other investigators, not on the opossum but on other animals. It would be a very extensive job, of course, to repeat such measurements in each of the opossum stages studied. The changes in the tunnel and pillars of Corti, during development, were the most striking features and the ones which appeared most closely associated with the hair-cells, for which reasons they were given primary attention.

With reference to the radial and other nerve fibers, we saw those in our Bodian sections in various stages, even the 48-day stage. We did not find it possible to trace them in any detail.

There is still a large amount of work to be done on this problem. Difficulties of getting adequate stages of material, making the tests, and then following up with adequate technic afterwards, are very great.

We hope to add to these observations, however, and present further details later on.

In Memoriam

Henry Bush Beeson.....Racine, Wisconsin
March 4, 1944

Oliver P. Bourbon.....Los Angeles, California
April 24, 1944

Ralph Stephen Chappell.....Indianapolis, Indiana
March 12, 1944

William Thornwall Davis.....Washington, D. C.
June 16, 1944

Carl Fisher .....Los Angeles, California
June, 1944

Raymond Willard Hawkins.....Rochester, New York
March 30, 1944

William Krieger .....Poughkeepsie, New York
May 19, 1944

Millen Alexander Nickle.....Clearwater, Florida
March 6, 1944

E. H. Porter.....Tiffin, Ohio
May 14, 1944

Raphael Silva .....Mexico City, Mexico
April 16, 1944

Charles M. Stiles.....Philadelphia, Pennsylvania
January 27, 1944

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Editorial

Industrial Ophthalmology

Industrial medicine has been evolutionary in its progress. Some years ago the practice of medicine in this field was incidental to the general practice without any facilities at the plants; whereas today industrial practice has become an important specialty, employing at the plant a staff of physicians, nurses and assistants, equipped with all the necessary facilities to render an almost complete medical and surgical service. When this special form of medical service was first proposed it met with considerable opposition both by some members of the medical profession and by employers. Today, however, most physicians and those concerned with management of industry admit that the extension of medical practice into this field has been of decided benefit to industry and to its employees. There is an increasing demand for the establishment of more medical staffs in industrial plants and for expansion in the personnel of these staffs so that a completely balanced service may be possible.

A better balanced service will be established when the importance of good vision in industry is recognized. It is evident that good vision is essential to efficient production, and the frequency of eye accidents is well known. However, the services of ophthalmologists as a component part of medical staffs have not generally been employed. Those interested in industrial vision and efficiency felt that this phase of industrial practice should be investigated.

With this object in view a Joint Committee on Industrial Ophthalmology was established, representing the Section on Ophthalmology of the A.M.A. and the American Academy of Ophthalmology and Otolaryngology. As an initial step this committee undertook an investigation to determine the justification for the belief that ophthalmic services in industry were in fact inadequate. The committee had as its objective a factual study of all specific existing factors relating to the vision of industrial employees and of ophthalmic services in industry. Where an inadequate service was clearly indicated the underlying causes were investigated, ways and means were studied and procedures were suggested for meeting the needs which such investigation revealed.

The committee has been motivated only by the purpose of discovering how the ophthalmologist may best use or contribute his special knowledge in the industrial field without any thought of "aggrandizing" the value of his specific services. On the contrary, the committee has been making a most thorough study of the shortcomings of ophthalmic practice in industry. It has devoted much time in disseminating information revealed by research and by clinical experience. It has given openly, freely and promptly by publication, lectures, discussion and seminars all data gathered so that anyone who can use this information may do so. Ophthalmologists have not been motivated by the thought of any selfish economic benefit. They do feel that it is their responsibility to promote a more adequate service in the industrial field, and that they must meet this responsibility in a sound and realistic manner.

The need for a better and broader service in industry and to its employees is generally recognized. However, some of the procedures which have been advocated to meet the need have been adversely criticized. For example, there has been criticism of the screening methods which are made by lay technicians — methods by which most defects in the vision of employees can be conveniently, quickly and economically discovered and recorded. There has also been criticism of the use of the scientific data gathered by them. Especially worthy of note is that made by Mr. C. O. Cozzens, vice-president of the American Optical Company, in a widely distributed pamphlet entitled "Industrial Visual Safety and Efficiency." Mr. Cozzens admits that:

"Industrial visual screening is essential and must be performed —"

"That adequate vision for the task to be performed should be an Industry 'must' —"

"That appropriate screening that determines the existence of an employee's deficiency — is essential to the needs and must be performed under the direction and supervision of the legalized refractionists."

We find that we are in agreement with these statements with the exception that we do not believe that the determination of employees' visual deficiencies, and all the problems relating thereto, can be completely and satisfactorily met by limiting ophthalmic services to any group professing knowledge and skill in refraction only. Such a group is not qualified to direct and supervise a complete ophthalmic service in industry. The refractionist can render a valuable service but it is limited. A trained technician can render a valuable service when such technician is skilled in making screening tests; and these tests will be most valuable when the data thus obtained is arranged in such a manner that most of the essential facts relating to the various visual functions of employees can be analyzed and coordinated with employee data. Superficial methods of screening have some though little value; the more complete the screening data the more useful it becomes.

Mr. Cozzens seems to fear that the more complete and modern screening methods that have been introduced during the past few years may create a "new group apart from the existing professions," and that this group will not be "responsible to the professions." He seems also to fear that should technicians perform screening that then a "complete corrective eye care" may be "vested in" a new non-professional group, separated and "uncontrolled" by professional responsibility, and that ultimately this group will become a "separate cult or fourth estate"; and finally that from this situation there will result a "dissipation of the benefits" of industrial eye care.

We confess that we are completely in the dark as to the grounds for these fears. Speaking for the ophthalmologists we have at no time advocated the establishment of a "new or separate cult" which might assume responsibility for eye care not legally permitted. The employment of assistants or of technicians is not a recent innovation; witness orthoptic technicians, the testing or screening of school children by teachers or by nurses, the testing of visual acuity by automobile licensing bureaus, and the supplying and fitting of goggles and of correcting lenses. The ophthalmologists have at no time advocated that lay technicians should assume the complete responsibility of eye care, free and separate from the supervision by the ophthalmologists. However, there are types of eye care which the non-professional man can perform. These services relate generally to the supplying and adjusting of protective goggles; the supplying, fitting or adjusting of corrective lenses (not prescribing); to the maintenance of protective equipment, and to the enforcement of safety rules. The ophthalmologist does believe that these services performed by non-medical technicians are essential to a complete and adequate ophthalmic service. Many of these services are no innovation and are used daily. We can find no valid objection to the employment of that type of screening service which gives valuable assistance to members of the medical staffs, to management, and to consulting ophthalmologists.

One procedure in this service, which is only a small part of the complete screening test, will eliminate as a rule 80 per cent of those who apparently have normal vision and who probably do not have visual defects requiring diagnosis, treatment or correction. Thus only 20 per cent would remain as referrals to be examined by those who are best qualified and who have legal authority to make the examinations. It seems obvious that in order to extend eye care to those who may need it, one must employ some reasonable method to discover the needy. Since screening methods can accomplish this object, they have been found to be most practicable and useful. In reference to the data secured or of services rendered by any screening method we know of none which can be separated from the other visual services. We know of no group foolish enough to contemplate a separatist movement. Cooperation not separation is desired by all who are interested in conducting screening tests and in compiling data on visual functions.

We are not directly interested in the "dissipation of the benefits" of eye care. We are interested in the visual welfare of industrial employees and we are conscious of our responsibility for supplying them with adequate eye care. We agree with Mr. Cozzens "that vision is a priceless treasure worthy of the best professional services." We believe that the best ophthalmic service can be rendered only by those having the best general and medical education and, in addition to this, a comprehensive knowledge covering the entire field of ophthalmology. Furthermore, special knowledge of the various industrial problems should be acquired by those desiring to practice industrial ophthalmology. However, anyone thus qualified to render an ophthalmic service will need assistance. He should not be expected to perform individually and unaided every detailed procedure which is necessary to a complex service in the field of industrial ophthalmology.

In the field of industrial ophthalmology there are problems which are common to general medical practice as well as to general ophthalmology. There are also problems or phases of practice which are peculiar to industrial production and the visual needs of industrial

employees. The ophthalmologist who can best meet these visual needs and services must have the cooperation of all groups whose services separately may be necessary to a complete ophthalmic service in industry. There can be no separate estate for any of these service groups.

Ophthalmologists have at no time advocated any separatist movement by any group interested in serving any of the ophthalmic needs of the public. On the contrary they have always held that complete cooperation with every group, which in their judgment could aid in any necessary part of such service, is essential to an adequate ophthalmic service.

Ophthalmologists as represented by the American Academy of Ophthalmology and Otolaryngology desire to fulfill their responsibility to industrial employees. They are searching for ways and means by which they can satisfy a desire on the part of the public for a wider dissemination of the benefits of their special knowledge in the industrial field. They desire the cooperation of all competent groups who can aid in the accomplishment of these objectives.

To accomplish these objectives, we believe that there should be further extension of industrial plant staffs, and that such staffs should include, at least as a consultant, an ophthalmologist who should be given the responsibility for the supervision of all ophthalmic services in industry.

Letter to the Editor

Dothan, Alabama
April 8, 1944

Dear Dr. Benedict:

Since you are Secretary of the Academy, I am writing this letter to you for your consideration and, if you find any virtue in the suggestion which it embodies, to pass it on to the Fellows of the Academy.

The suggestion is this:

There are undoubtedly a good many young men and boys in the armed services who are the sons of Fellows of the Academy and who, in many cases, find themselves in a strange place and among strangers.

It has occured to me that if these boys were told by their fathers or if their fathers would communicate wth members of the Academy in the region in which the youngster found himself, then we might be able to look up these boys and see that they were making out all right.

Since I live in the deep South and since the air corps is training in this section, largely, I have often wondered if there were not some boy in this section who sort of needed a cheering up from some man or family of his own kind.

If you think anything of this suggestion then I might further suggest that it be put in operation that the fathers or boys themselves notify such members of the Academy as reside in the region in which their son finds himself so that those boys might be contacted and entertained by men and families who are essentially of their own kind.

It would seem to me that medical men should draw very close to each other these days and that this little thing might be instrumental in giving comfort to some distraught mother or father whose son is far from home—particularly since veritable children are inducted into the service.

With kindest regards to you I am,

Sincerely yours,

JOHN A. KEYTON

Committee Activities

COMMITTEE ON STANDARDIZATION OF TONOMETERS

Report on Arrangements for Making Certified Tonometers Available to the Profession

The problem of providing the profession with reasonably accurate tonometers is twofold. In the first place we have to deal with the several thousand instruments which are, so to speak, out in practice. Many of these instruments, though sold as "Schiötz" tonometers, actually differ very seriously from the original standard instrument designed and calibrated by Schiötz. Instruments of grossly defective construction have been produced in recent years both by American and by foreign manufacturers. Even many of the instruments produced in Oslo during the prewar period, and certified by A. Tandberg, the assistant to the inventor, have been found to differ very considerably from earlier models certified by Schiötz. It is no wonder with the measuring instruments showing such large variations in their essential physical characteristics that wide variations in clinical estimates of the intraocular pressure in individual cases have resulted.

In order to assist the practicing ophthalmologist in determining whether or not the instrument which he is using is a reliable one, tonometer checking stations have been established in New York, Chicago and San Francisco to which instruments can be sent for examination. The work of these checking stations has been previously reported.[1]

There is no reason, however, why the profession should continue to accept instruments which vary in this uncontrolled fashion in respect toessential features of their design. In order to eliminate these variations from current and future production, the Committee on Standardization of Tonometers has for the past year been negotiating with American manufacturers of tonometers with a view to establishing fixed standards which will be complied with in the manufacture of Schiötz tonometers in this country. Only those instruments which conform to Schiötz original specifications within reasonable limits can properly be used to measure the intraocular pressure with the aid of the Schiötz calibrating scale.

Such agreement on what is a standard Schiötz instrument does not preclude the possibility that instruments of other and improved design may be developed by the profession and the manufacturers, but if an instrument is to have a plunger of different weight and diameter from that calibrated by Schiötz, it cannot be expected to give the same scale readings as does the standard Schiötz instrument, and the Schiötz calibrating chart is not applicable to such an instrument.

All of the American manufacturers of Schiötz tonometers have now agreed to produce instruments which are to

conform in respect to the essential features to standards mutually agreed upon. These standards are based upon the original specifications of Schiötz.

In order to be sure that the instruments do, in fact, correspond to the specifications that have been agreed to, the Committee has felt that an independent measurement of the instruments before their sale should be provided. We, therefore, have arranged with the Electrical Testing Laboratories, Inc., of New York City to weigh and measure the instruments submitted to them for testing by the various manufacturers. Those instruments which are actually found to conform to the agreed specifications will be certified as such by the testing laboratory. The form of certification which the testing laboratory has been authorized to issue is the following:

ELECTRICAL TESTING
LABORATORIES, INC.
2 East End Avenue at 79th Street
New York 21, N. Y.

CERTIFICATE OF
INSPECTION AND TEST

Schiötz Tonometer No. .....
Manufactured by .....
was inspected and tested on.....
and found to comply with Specification No.
.....for Schiötz tonometers.
These specifications are approved by the Committee on Standardization of Tonometers of the American Academy of Ophthalmology and Otolaryngology.

Specifications supplied upon application.
Electrical Testing Laboratories, Inc.

.....
Head, Photometric Division

Inspected and Tested by .....
Date.....

.....
E. L. T. No.....

The Electrical Testing Laboratories, Inc., has agreed to submit an annual report of its work in this field to the Committee on Standardization of Tonometers, and to keep the record of its tests available for the Committee's inspection and study. The Electrical Testing Laboratories, Inc., is a commercial organization and will charge the manufacturer a fee for the testing of his product. The Committee has no responsibility for the size of the fee charged, but the fee will be the same for all manufacturers. Present estimates indicate that the manufacturers will be able to sell certified instruments to the profession at an additional charge of only about $2.50 over the cost of uncertified instruments.

The arrangements for certification of Schiötz tonometers described above were submitted to the Council of the American Academy of Ophthalmology and Otolaryngology before they were finally agreed to, and have been approved in principle by the Council.

The manufacturers of Schiötz tonometers who have agreed to submit their product to the Electrical Testing Laboratories for testing and certification are Sklar, Pilling and Meyrowitz. Instruments produced by these manufacturers and certified by the Electrical Testing Laboratories, Inc., can be accepted by the profession as conforming within reasonably narrow limits to the original reference standard instrument of Schiötz. At the present time, the Committee on Standardization of Tonometers is engaged in working out similar arrangements for the certification of Gradle-Schiötz Tonometers.

SPECIFICATIONS FOR STANDARD SCHIÖTZ TONOMETERS

The specifications which have been agreed to were reached after a prolonged period of analysis and negotiations. The first step in this direction was taken by Drs. Schoenberg and Posner[2] from an analysis of the original description of Schiötz. These specifications were then used as a basis for the measurements made at the tonometer checking stations by Drs. Schoenberg and Posner in New York, and by Dr. Kronfeld in Chicago. Methods of measurement were worked out at these stations and also with the collaboration of Mr. William F. Little of the Electrical Testing Laboratories who has given most generously of his time and effort in this matter. Much help in this respect was obtained also from Mr. Raymond Mehrlust, engineer in charge of the development of the Sklar tonometer. Dr. Kronfeld then undertook a special study[3] to find out whether the measurements initially decided on actually included those most significantly related to the clinical efficiency and accuracy of the instrument. At the same time, Mr. Little made measurements on a batch of 50 instruments supplied him by one of the manufacturers, and the data which he obtained were analyzed in order to discover how large were the variations to be expected from instrument to instrument under current manufacturing practice.

Based on these analyses a preliminary draft of specifications, tolerances, and methods of measurement was drawn up by Drs. Friedenwald, Kronfeld and Schoenberg in collaboration with Mr. Little, and submitted to the Committee on Standardization of Tonometers, and also to the manufacturers of tonometers for comment and approval. The specifications agreed to as the result of these discussions are listed below. The Committee, however, does not regard these specifications as ultimate and final. Extensive clinical trials will be required to disclose whether instruments which conform to these specifications do in fact give accurate and comparable readings on human eyes. Such clinical experience may indicate that the allowed tolerances are too great and that further sharpening of the specifications may be necessary. With experience the manufacturers may also be able to improve on current methods of manufacture. Consequently the Committee proposes to review the specifications from time to time in the hope of making them still more rigorous. Even with the currently accepted specifications, however, the Committee is confident that a very great improvement in the uniformity and reliability of Schiötz tonometers will be achieved.

REFERENCES

1. Committee on Standardization of Tonometers. Report on tonometer checking stations. Transactions Amer. Academy of Ophthalmology and Otolaryngology. Jan.-Feb., 1944.
2. Schoenberg, Mark J.,and Posner, Adolph: A report on defects found in tonometers examined at the checking station of the National Society for Prevention of Blindness, Am. J. Ophth. 27:368 (April) 1944.
3. To be reported elsewhere.

SPECIFICATIONS FOR SCHIÖTZ TONOMETERS

All tonometers are to carry a serial number. The manufacturer of the tonometer is to be designated on the tonometer

Test	*Specification* *Standard*	*Limits*
Weight, grammes		
Plunger, lever and pointer		
Scale indication 5	5.5	±0.15
Scale indication 10	5.5	±0.25
Tonometer without handle	16.5	±0.5
Load 7.5	2.0	±0.02
Load 10.0	4.5	±0.02
Friction between plunger and sleeve	Negligible	
Friction between handle and sleeve	Negligible	
Foot plate, millimeters		
Diameter	10.1	±0.2
Curvature	15.0	±0.25
Diameter of curved surface	9.0 minimum	
Plunger, millimeters		
Diameter (measured 0°-90°)	3.0	±0.03
Curvature	15.0	± .75
Edge curvature — smooth, rounded		0.1 maximum
Scale indication		
On 15 mm. standard	−1	±0.1
On 16 mm. standard	0	±0.1
On own block	0	±0.1
Micrometer		
Scale 5	5.0	±0.25
Scale 10	10.0	±0.25
Scale 18	18	±1.0
Wobble		
Plunger	0	0.4 maximum
Fulcrum	0	0.4 maximum
Extension of plunger below foot plate, millimeters		
Pointer, millimeters		
Depth	...	3.0 maximum
Thickness	...	0.25 maximum
Distance between scale and pointer	...	1.0 maximum
Plunger contact with hammer	Perpendicular 5-10 scale	
Hammer, underside smooth, free from pits		
Schiötz Chart correct		

FRANCIS H. ADLER, M.D.
FREDERICK C. CORDES, M.D.
HARRY S. GRADLE, M.D.
PETER KRONFELD, M.D.
JOHN MCLEAN, M.D.
ADOLPH POSNER, M.D.
MARK J. SCHOENBERG, M.D.
JONAS S. FRIEDENWALD, M.D., *Chairman*

ROLE OF CILIARY ACTION IN PRODUCTION OF PULMONARY ATELECTASIS. VACUUM IN PARANASAL SINUSES, AND IN OTITIS MEDIA

A. C. HILDING, M.D.
DULUTH, MINNESOTA

IT WAS my privilege last year to give a paper at this meeting on the effects of loss of ciliary function especially in asthma and influenza. Strange as it may seem, there are other pathological situations in both the upper and the lower respiratory tract which seemingly do not occur unless the cilia are functioning actively. Among these are vacuum headache, postoperative pulmonary atelectasis, and probably retraction of the ear drum.

Last spring, one entire sesion of the weekly clinical pathological conference in our city was given over to the discussion of the development of postoperative pulmonary atelectasis. The literature was reviewed, and there was much excellent discussion by surgeons, internists, endoscopists, and pathologists. But there was no mention of ciliary activity in the entire discussion. It seemed to me that the ciliary mechanism must be involved, and I decided to try to determine in what manner.

The literature on the subject of atelectasis is voluminous, and extends back a hundred years. Coryllos and Birnbaum,[1] who have done much work on atelectasis, conclude that there is only one cause, namely, "bronchial obstruction with absorption of the air distal to the obstruction." They speak of "firmly fixed bronchial plugs" of secretion as the usual cause of obstruction. This, in fact, seems to be the belief of most modern writers.

De Takats, Fenn and Jenkinson,[3] writing just last year, agree substantially that the cause is obstruction by massive secretion. "Weakened respiratory force with accumulation of mucus from insufficient movements of cilia and suppressed cough are cause enough." They cite experiments in which bronchial constriction and production of excess mucus were produced reflexly by chest injury and visceral manipulation.

Van Allen and Adams[9] attempted to demonstrate the mechanism of atelectasis in experiments on 51 dogs. They destroyed a ring of bronchial epithelium through a bronchoscope by application of a strong solution of silver nitrate. From these experiments they made the following conclusions: atelectasis did not develop in a normal lung during quiet respiration, but was favored by strained respiration and cough; a valvular obstruction produced it faster than complete obstruction; the pent-up air was probably absorbed by the blood. Their conclusion, concerning the action of deep respiration, is at variance with clinical experience which seems to indicate that deep respiration and cough prevent atelectasis. They made another observation which seems odd. Some of their dogs developed a stricture with complete closure of the treated bronchus. The lung tissue behind these closed strictures did not become atelectalic, but floated when placed in water.

Presented at the Forty-Eighth Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 10-13, 1943, Chicago, Illinois.

This work was aided by a financial grant from Dr. D. L. Tilderquist, associate of Dr. A. C. Hilding.

Lee, Tucker, and Clerf[7] produced atelectasis in dogs by instilling into the bronchi some secretion which they had aspirated from the bronchi of a patient suffering from atelectasis.

After reading a number of these articles, one is left with several mental conflicts. Why should deep breathing prevent atelectasis in patients, and produce it in dogs? If obstruction is the cause of atelectasis, why was there no atelectasis in the dogs in which the bronchi were completely closed by scar? Why do not asthmatic patients, who die of suffocation because their bronchi are so completely filled with heavy mucus, develop atelectasis? If osbtruction in atelectasis is caused by reduced ciliary activity, why did the dog of Lee, Tucker and Clerf develop atelectasis? Presumably, that dog's ciliary mechanism was normal. Ciliated epithelium readily carries away large loads of tenacious mucus without any difficulty; in fact, heavy tenacious mucus seems to be carried better than any other type.[5] Why wasn't it removed from this dog? What "fixes" these mucinous "plugs" so firmly in the bronchi? How can they become "fixed" when the entire bronchial lining, so to speak, is moving upward? One might as well speak of an object on a moving conveyor belt, or escalator, as being "fixed." These mucous "plugs" do not appear at necropsy to be "fixed." They simply look like a quantity of secretion lying upon the epithelium. They cannot be "fixed" upon ciliated epithelium — yet, there they are.

Asher, the Swiss physiologist, once said in a lecture, "When two observers bring in seemingly contradictory reports, there are several possibilities to consider: The first one may be right, and the second one wrong; or, the first one may be wrong, and the second right; or both may be wrong; or both may be right."

May we review briefly some rudimentary facts of physiology which seem to bear upon this subject? When the size of the thoracic cavity is increased by inspiratory effort, a region of reduced air pressure is produced. Since this region is open to the out-of-doors, atmospheric pressure forces enough air into the chest to bring again the pressure up to that of the atmosphere. The air flows down through the trachea, bronchi, and bronchioles until it reaches the dead ends in the alveoli. These it causes to distend. The trachea and bronchi increase in diameter and length, and the lungs slide down the parietal pleura toward the abdomen. Expiration is accomplished by increasing the pressure within the thorax. Several factors enter into this procedure, such as the elasticity of the lungs, the weight of the relaxed chest wall, release of the abdominal organs by the diaphragm, and, when necessary, voluntary muscular contraction. The contained air flows out toward a lower pressure. The lungs rise and contract, and the air passages shorten and become narrower. The movements of the lungs and air tubes are purely passive. The volume of the lung at expiration may be readily doubled or trebled by deep inspiration. The diameter of the bronchi is said to vary as much as a third.[4] Consider for a moment what the reduction in diameter of a cylinder by one-third means in terms of volume. A cylinder measuring 10 cm. in length, and 3 cm. in diameter, contains about 71 cc. If it is reduced in length by one-fifth, and in diameter by one-third, the volume is reduced to about 25 cc., or, by approximately two-thirds.

The normal respiratory tract is lined with a thin film of mucus throughout its entire extent. This entire lining film is removed by ciliary action perhaps one to three times each hour, and as often replaced by the mucus glands. The total volume of mucus normally present is extremely small since the film is vanishingly thin. It has been necessary in the

course of some of these experiments to obtain small quantities of respiratory mucus. It required 5 decapitated hens to produce about .75 cc. The entire respiratory tracts of 12 freshly killed pigs, when hung up for about an hour, furnished at the larynx a total of between 1 and 2 cc. In the same manner it was possible to obtain about 1 cc. from the respiratory tracts of 6 cows.

There are some physical characteristics of this respiratory mucus which should be mentioned. It tends to span across spaces — forming bubbles, diaphragms, and threads. It has a certain tensile quality, and can be dragged for a considerable distance. The cilia transport it readily, and can seemingly carry a hundred times the normal load without difficulty. (Fig. I.)

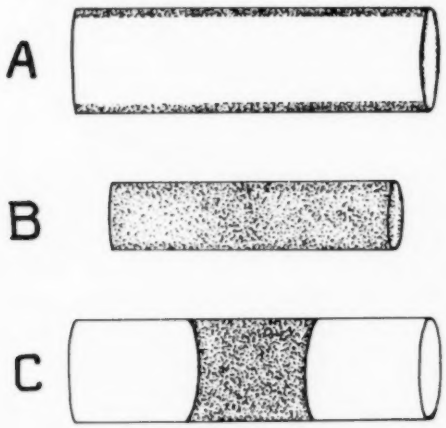

FIG. I

Diagramatic representation of how mucus might span a bronchus due to changes in the length and diameter of the bronchus during respiration. Cylinders A & C measure 3 cm. in diameter and 10 cm. in length, and contain a volume of about 71 cc. Cylinder B is one-fifth shorter, and one-third narrower, and contains only 25 cc. If cylinder A, containing a lining of mucus as indicated by the stippling, should be reduced in size to that of B, and then reexpanded to its original size C, the mucus would fill B, and take a shape and position approximately as shown by the stippling in C. Something similar to this, undoubtedly, takes place in the bronchi when an excessive amount of mucus is present.

A possible explanation of the development of atelectasis occurred to me, which, if true, would resolve the apparent conflicts that we have mentioned. Consequently, I spent a few days in a laboratory* in order to test this possibility. After some fruitless experiments with commercial bovine mucus, we finally found something that seemed to be significant.

EXPERIMENT

A short section of trachea, taken from a freshly killed hen, was pinned horizontally to a small board, and a mass of mucus that had been collected from the respiratory tracts of several hens was introduced into the lower end. It was sufficient in size to occlude the lumen completely. It moved by ciliary action from the lower to the upper end in a few minutes. The mucus was then recovered, and again introduced into the lower end. As soon as it began to move, the trachea behind it was stoppered and connected with a small water manometer as in fig. II. Almost before the stopper could be secured, a negative pressure began to develop. (Fig. III.) It reached −34 mm. of water in about eighteen minutes.

This experiment was repeated several times with the same result each time. A negative pressure developed behind the moving mass of mucus as the latter advanced. The further the mucus advanced the greater the negative pressure, and the greater the negative pressure the slower the progress of the mass. Finally it would come to a complete stop. The greatest negative pressure recorded was about 35 mm. of water.

This simple experiment seems to be the key to the development of atelectasis. It is essentially a piston-cylinder action. The mucous mass, motivated by cilia, acted as a piston moving within the cylindrical piece of trachea. It formed an air-tight seal and moved along until it was stopped by air pressure. It came to

* These experiments were done at the University of Wisconsin, in the Department of Pathology, where Acting Dean Walter J. Meek very kindly made laboratory facilities with assistance available to me.

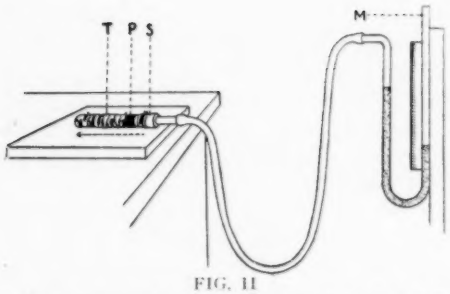

FIG. II

Arrangement of section of excised trachea (T) from a freshly killed hen, and the water manometer (M), in the experiments demonstrating that a negative pressure is produced by ciliary action. The lower end of the trachea was stoppered and connected with a manometer while the upper, or laryngeal end, remained open. A mass of mucus (P), sufficiently large to occlude the lumen, was first introduced into the lower end of the trachea, and as soon as ciliary action had started it on its journey toward the other end, the stopper (S) on the manometer tube was introduced and secured. The manometer indicated a reduction in pressure almost immediately.

A control experiment was done in which the position of the tracheal section was reversed. The stopper and manometer tube were placed in the upper or laryngeal end and the mucus again introduced into the lower end. This time a positive pressure developed promptly (see fig. VII).

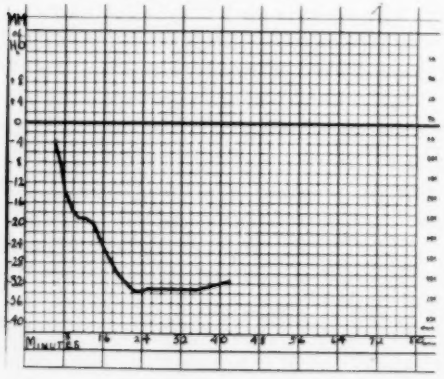

FIG. III

Curve showing the fall in pressure in the closed space behind a mass of moving mucus in the section of hen's trachea illustrated in fig. II. Ciliary action moved the mucus against atmospheric pressure while the pressure in the space behind the mucus fell. The figures on the ordinate indicate pressure in mm. of water, while those on the abscissa indicate time in minutes. The pressure fell 34 mm. of water in 18 minutes.

a standstill, but was not in any other sense "fixed." The cilia can move large masses of mucus with no difficulty, but they cannot lift 15 pounds of atmospheric pressure with a piston of slippery mucus. When the pressure in front of the advancing mucous piston, minus the pressure behind it, became equal to the effective force which the cilia were able to apply, the "piston" of mucus became "stalled."

One experiment was continued for fifty minutes. (Fig. IV.) The negative pressure in the manometer was maintained at maximum for eight or ten minutes, and then began to decrease. It finally returned again to –6 mm. from a maximum of –14 mm. Meanwhile, the mucus began to appear at the distal end of the trachea where it formed a thick ring. We removed the stopper, supposing that all of the mucus had already been transported, and that the pressure of –6 mm., which still remained, must be an effect of capillarity. The water in the manometer promptly equalized. When the trachea was inspected, it was found that a very thin transparent diaphragm of mucus was still intact. This had ap-

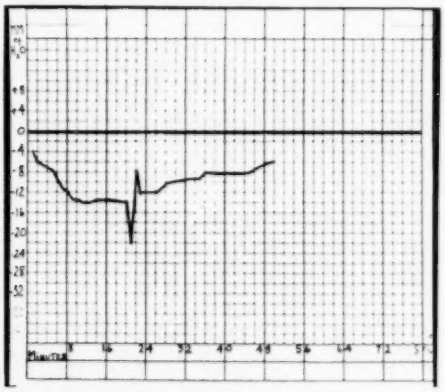

FIG. IV

Curve showing the fall in a hen's trachea behind a mass of mucus moved by ciliary action. After 22 minutes the pressure began to rise again, and after 50 minutes had returned practically to atmospheric pressure. Fig. V indicates the changes in the mass of mucus which caused the pressure to rise again. The sudden, marked changes in pressure, indicated by the two sharp points, occurred when the position of the trachea was changed from horizontal to vertical. At 21 minutes the trachea was held vertically with the laryngeal end down, and with the cilia working in the direction of gravity. The pressure was noted and found to have fallen 8 mm. more. At 22 minutes the trachea was held with the laryngeal end up, and with the cilia working against gravity. The pressure rose 5 to 6 mm.

parently maintained the small pressure of –6 mm.

The cilia obviously had not stopped working when the mucinous piston came to a standstill. They continued to exert traction with the result that mucus was dragged away at the periphery and carried progressively toward the distal end of the trachea in a thin film. (Fig. V.) The volume of the piston was reduced in this manner, thereby also reducing its thickness and strength. As it became thinner, it could no longer maintain the maximum pressure, and sagged backward in the middle, thus reducing the negative pressure. This continued progressively until all of the mucus had collected in a thick ring at the distal cut end of the trachea, and the negative pressure had practically disappeared.

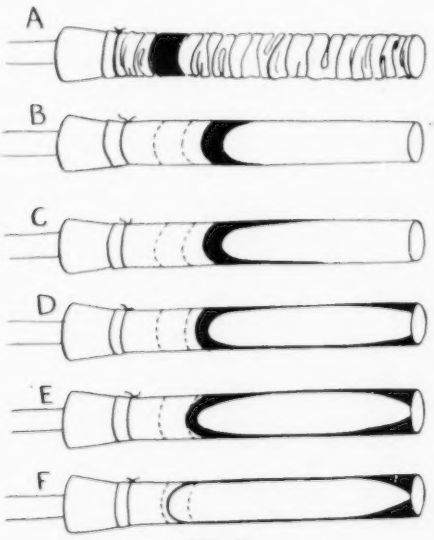

FIG. V

A diagnostic representation of the changes in the shape and position of the mucus mass during the pressure change shown in fig. IV. A shows the position when the stopper was introduced. In B it had advanced to the "stalling" point. In C the margins were advancing by ciliary action, while the main mass remained stationary, held by atmospheric pressure. D, E, and F show progressively advance of the mucus in thin films to the cut end of the trachea where it collected in a ring. Meanwhile, the occluding diaphragm grew progressively thinner and weaker as its volume was reduced, and sagged back toward the starting position, thus causing the pressure behind it to rise again.

The dotted lines in each sketch represent the position when the stopper was introduced.

It now seems possible to reconstruct the steps in the production of atelectasis in a surgical patient whose respiratory tract is normal. The principles here outlined doubtless apply also in other forms of atelectasis. (Fig. VI.) First, an abnormally large quantity of mucinous secretion is produced. This may be due to the anesthetic, reflex stimulation, or a combination of these and other causes. Second, the secretion occludes the lumen of the air passages by spanning across it. The following circumstances combine to cause this occlusion: force of inspiratory flow of air, changes in diameter and length of the bronchus, bellows-like action of the involved lobe, and the viscosity and volume of secretion. Third, the occluding mucinous mass advances up the bronchus, causing a slightly reduced pressure in the lobe. Fourth, the elastic tissue in the lobe reduces the size of the lobe as the pressure falls. Fifth, the alveoli of neighboring lobes are carried forward by inspiratory air into the space which is relinquished by the affected lobe. The sliding movements of the lungs during the respiration facilitate the shrinkage of the affected lobe, and the enlargement of the neighboring lobes.

These steps would be repeated many times. One mucinous piston advancing up the bronchus would not produce collapse. There would have to be a succession of them, each carrying along a volume of air somewhat on the principle of a mercury vacuum pump. Each one would eventually reach a point where the increased diameter of the air passages, and the increased force of impact of inflowing air, would rupture the diaphragm across the lumen, and liberate the bubble of air behind it. The mucus would continue its journey as a mural mass or film. The pistons would continue to advance and be carried away as long

as any considerable amount of air remained in the lobe. They would not come to a standstill until the pressure within the lobe had fallen to such an extent that the effective power of the cilia had been overcome. This would not occur until the air in the lobe was exhausted, and the lobe had collapsed.

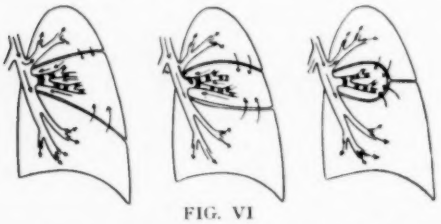

FIG. VI

Diagram representing the various steps in the production of postoperative pulmonary atelectasis. The middle lobe is the affected one. (1) An excess of secretion is formed in the lobe. (2) A series of occluding masses, or pistons of mucus, form across the lumen. A combination of circumstances enter into their formation, such as: force of inspired air, changes in diameter of the air passages, and so on. (3) The "pistons" move up the cylindrical air passages by ciliary action, each carrying a quantity of air. (4) As soon as the air pressure within the lobe begins to fall, the lobe shrinks by its own elasticity, and by pressure, from surrounding lobes. (5) The surrounding lobes carried by the force of inspired air move into the space relinquished by the affected one. The changes in position of the lobes, as well as their shrinkage and distension, are facilitated by the sliding motion of the lungs during respiration. (6) The advancing "pistons" of mucus rupture serially as they reach tubes of greater diameter, and meet more forceful changes of air pressure. They then release the air bubbles which they have carried, and continue on their course as mural films or masses. (7) Production of considerable negative pressure in the affected lobe so great that (8) the mucous masses then present in the air passages come to a standstill against atmospheric pressure. These masses form the so-called "firmly-fixed mucous plugs" referred to in the literature.

The seemingly conflicting observations can now be resolved. All of the observers were correct in their observations. The experimental atelectasis of Van Allen and Adams is not comparable to postoperative atelectasis because they destroyed the continuity of the ciliary mechanism, thus materially altering the conditions of the experiment. The same can be said concerning the dogs in which the bronchi became completely occluded by scar. The masses of mucus which were supposedly "firmly fixed" in the bronchi are not fixed at all. They simply have been stopped by a column of air against which the cilia cannot advance them. Given time the cilia may drag the secretion around and past this obstruction.

It has been assumed that the air entrapped behind the mucous plug is absorbed. Undoubtedly absorption does remove some of it—perhaps most of it—but the piston-cylinder action just described very likely plays an important role also.

EXPERIMENT

There remained a conceivable possibility that the air in the excised trachea used in the above experiment might have been absorbed by the tracheal tissue, thus producing a reduced pressure. To determine this, another experiment was done reversing the direction of the section of the trachea. That is, the upper or laryngeal end was stoppered and connected with a manometer. When the

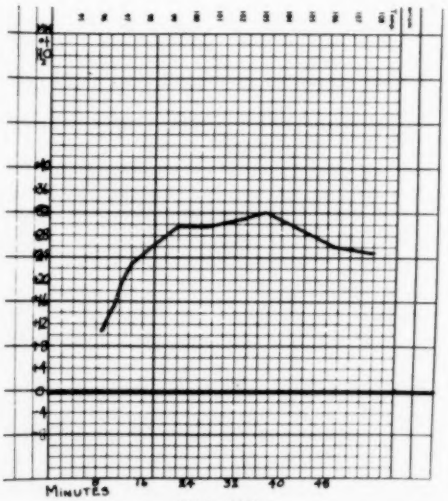

FIG. VII

Curve showing positive pressure produced in a section of excised trachea from a freshly killed hen, when the trachea was reversed in position. An occluding mass of mucus was introduced into the lower end, and the upper, or laryngeal end, stoppered, and connected with the manometer. This shows that the negative pressure found in the experiments illustrated in fig. II and fig. III was not due to absorption of air by the tissues.

mucus was introduced at the lower end, and carried upward by ciliary action, a positive instead of a negative pressure promptly developed. (Fig. VII.) This pressure reached + 32 mm. of water and was maintained near that level for over half an hour. It did not go down, apparently because the mucus was carried against the stopper, and also against the end of the manometer tube, and held there by ciliary action. This experiment shows that the negative pressure developed in the experiment was not due to absorption. No one would seriously argue that the air was absorbed behind the mass of mucus and excreted again in front of it.

The question arises as to whether the ciliary mechanism can maintain the large negative pressures that are found clinically in cases of massive collapse of the lung (up to 600 mm. of water. Davies[2]). No direct observations as to that have as yet been made. But if a half cubic centimeter of mucus in a short section of excised hen's trachea, and covering only a few square millimeters of epithelium can sustain a pressure of 35 mm. of water, it is not at all unlikely that an ounce or two of secretion distributed through an entire bronchial tree might sustain a pressure 10 or 20 times as great. If this is not true, then it is difficult to imagine what power does hold the mucus suspended thus between atmospheric pressure and a pressure of 600 mm. lower. It is hardly likely that the viscosity of the secretion could hold it because this type of secretion can be put through a 16 or 18 gauge hypodermic needle without any difficulty.

The production of vacuum by ciliary action has applications in other fields which are of more immediate interest to most of us, namely, in the sinuses and in the middle ear.

The occurrence of headaches due to reduced pressure in the sinuses has been accepted by rhinologists for many years. Yet there has been no good evidence to support the theory, and more recently there has been a tendency to discredit it for that reason. Proetz[8] gives Sluder credit for first popularizing the theory, but states that it was based only on Sluder's personal belief. He discusses the physics of likely gaseous exchange in a closed sinus, and concludes that on this basis a slight positive pressure, but no vacuum, might develop. He mentions as a possibility that a vacuum might develop when swollen mucous membranes within a sinus become depleted through their vascular channels in the presence of a blocked sinus.

There can be no doubt that a certain peculiar type of headache develops in sinusitis which may often be relieved by shrinkage of the mucosa near the ostium. It occurs after the purulent stage when the secretion is becoming heavy and mucinous. To the patient, it is an exasperating and demoralizing pain that extends into his teeth and behind the eye. Suddenly he feels, and may even hear, the leakage of air through a narrow chink somewhere, and the pain is miraculously gone. A few moments later, he may spit out a large mass of viscid mucus.

The findings of the piston-cylinder action in the trachea of the hen suggested the possibility that a similar mechanism might produce negative pressure in the sinuses. With the help of Dr. Hiram Essex, of the Mayo Clinic,* we did a few successful experiments that seem to be conclusive.[6]

EXPERIMENT

A dog was anesthetized with ether by the open drop method. Considerable secretion gathered in the trachea from whence it was collected. This mucus was used to inject directly into the frontal sinuses of another dog.

* The officers of the Mayo Foundation and Dr. Frank Mann have very kindly permitted me to use the laboratories at the Institute of Experimental Medicine and have furnished materials gratis.

TECHNIC

The dog was anesthetized with ether, and placed in the prone position. Two small holes were made through the roof of the left frontal sinus, and one through the roof of the right by means of a punch. The holes were of such size that a gauge 18 hypodermic needle fitted tightly in them. One needle was placed in each of the three holes. The needle in the right frontal sinus was connected to a small water manometer, and one of the needles in the left frontal sinus was connected to another water manometer. The right sinus served as control. (Fig. VIII.)

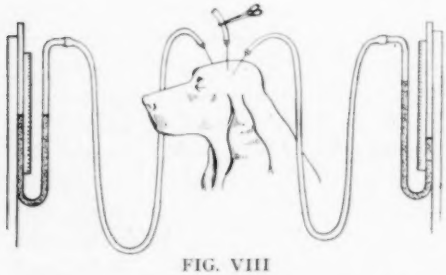

FIG. VIII

Arrangement of needles and manometers in the experiments on the frontal sinus of the dog. Mucus was injected into the left frontal sinus. A negative pressure developed as the mucus was removed by ciliary action. The right sinus served as control, and showed no change of pressure.

A quantity of mucus (3 to 7 cc.) was injected into the left frontal sinus through the second needle, and the needle promptly closed off. A negative pressure, as indicated on the manometer, began to develop in the left frontal sinus after a few minutes. The maximum recorded was –66 mm. of water. (Fig. IX.) There was no pressure change noted in the control sinus in any experiment.

The experiment was repeated 3 times. The duration of the first experiment was one hour; of the second, two hours; and the third, four hours and a half. The first failed to show change of pressure. The sinus was opened at the end of the experiment and was found to contain a very large ostium which had apparently not become occluded. The other 2 experiments were successful and gave similar results. Negative pressure began to show in 1 after five minutes, and in the other after seven minutes. Both showed an initial positive pressure, 1 of 15 mm. of water, and the other 270 mm. The maximum negative pressure was reached in about twenty-five minutes in both. The negative pressure was maintained at or near maximum for twenty minutes in 1, and thirty-four minutes in the other, after which the pressure began to recede. Recession of negative pressure was slow and incomplete in both, according to the manometer readings. In 1, it receded 13 mm. (i.e., the pressure in the sinus rose 13 mm.) in one and one-fourth hours, and in the other it receded 23 mm. in three and one-half hours.

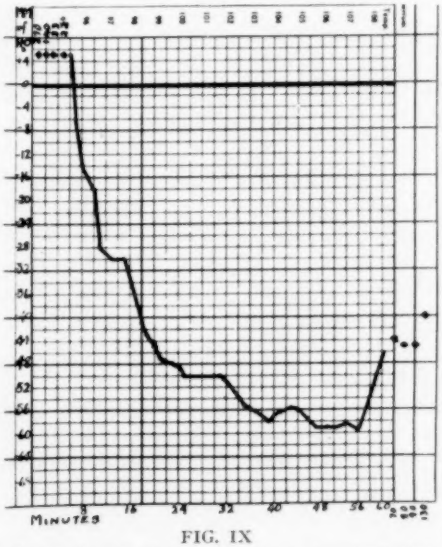

FIG. IX

Curve showing the negative pressures obtained in the left frontal sinus of dogs after the injection of a few cc. of mucus.

The sinus used in the longer experiment was opened at the end of four and one-half hours. It was practically empty. The end of the needle connected with the manometer was found to be plugged,

and the reading in the manometer remained unchanged even after the sinus had been opened. When the needle was removed from the tubing, the water surfaces in the manometer promptly leveled.

The findings in the sinus experiments were somewhat different from those in the experiments on the trachea. There was an initial positive pressure, the fall in pressure was greater, and the return to a normal pressure reading failed to occur. The initial positive pressure was doubtless due to the fact that the ostium became covered with mucus and occluded before the entire amount had been injected. The remainder had to be forced in under pressure. The greater fall in pressure was doubtless due to a combination of circumstances, such as the larger amounts of mucus used, the difference in the shape of the cavities, and the fact that the experiments were done on the intact living animal, whereas excised tissue had been used in the experiments on the trachea.

The failure of the pressure to return to normal was merely apparent. As soon as the pressure began to rise, secretion entered the manometer needle and blocked it. The reason for this is the fact that the ciliary flow from some parts of a dog's frontal sinus to the ostium is routed across the roof, and a needle protruding through the roof is almost certain to be covered with secretion, if any considerable amount is present. This would not affect the manometer reading materially, as long as the pressure in the sinus was falling, and air was passing through the needle into the sinus. However, as soon as the pressure in the sinus would rise above that in the manometer, then air would pass through the needle into the manometer, and would carry secretion with it.

Clinically, vacuum in an infected maxillary sinus would apparently develop as follows: (1) The air contained in the sinus at the beginning of the attack would pass out through the ostium as the cavity filled with initial watery secretion, and remain devoid of air until the attack was nearly over. (2) The various types of secretion (watery, purulent, and mucinous) would pass through the ostium during the course of the attack by a combination of forces, such as gravity, ciliary action, and pressure from continuous production of secretion. (3) The production of secretion eventually would drop below the capacity of the cilia to remove it. This would be during the late stages when the secretion is mucinous. (4) The traction exerted upon the contained mucus, by the cilia in the nose, in the ostium and in the sinus, would cause a fall in pressure that would be abolished only when air could force its way past the secretion at the ostium and into the sinus. (5) Despite the negative pressure in the sinus, secretion would continue to leave the sinus as the cilia removed thin films from the sides of the contained mass. (6) Eventually the portion of the mucinous mass remaining within the sinus would be small enough to permit air to pass by through the ostium.

Several attempts have been made to produce vacuum in the middle ear by injecting mucus into the hypotympanum and first part of the eustachian tube. To date they have not been successful. The ears of pigs and cows have been found to be unsuitable, and in my only attempt at necropsy the body was too cold. The cilia had ceased to act.

Nevertheless, I feel sure that at least under certain circumstances the action of cilia moving masses of mucous down the eustachian tube produces the negative pressure. We know from the retraction of the ear drum that negative pressure frequently occurs in the middle ear.

It is, of course, quite possible in all of these conditions that absorption of air by the bloodstream may play a role.

The treatment of atelectasis seems almost self evident. Prophylaxis must seek to avoid the formation of occluding masses of mucus. All possible measures should be taken to avoid the production of excess secretion. If abnormal quantities of mucus do form, then deep respiration and frequent change of position would tend to prevent the formation of occluding masses. When atelectasis is already present, measures must be taken to replace the air in the collapsed lobe. Deep breathing may force air past or through the occluding masses. If not, then either aspiration of the mucus or inflation would be indicated.

SUMMARY

The steps in the development of postoperative pulmonary atelectasis are as follows: (1) An excess of secretion is formed within the affected lobe. (2) A succession of occluding masses, or "pistons," of mucus form across the lumina of the air passages. (3) These "pistons" move up the cylindrical air passages by ciliary action, each carrying before it a quantity of air. (4) As soon as the pressure within the lobe begins to fall, it shrinks by its own elasticity, and from pressure by adjacent lobes. (5) The adjacent lobes carried by the force of inspired air, move into the space relinquished by the affected one. (6) The advancing pistons rupture serially as they reach tubes of greater diameter, and meet more forceful changes of air pressure. Each then releases the bubble of air which it carries, and continues on its course as a mural mass of film. (7) A negative pressure of considerable proportion is produced within the lobe when the supply of air is exhausted. (8) The masses of secretion then present in the air passages come to a standstill when the cilia can no longer advance them against atmospheric pressure. (9) The cilia continue to remove secretion from these "stalled" masses in thin films, and might eventually remove them entirely if they were not replaced by continued secretion.

Negative pressure can be produced within a normal sinus by the introduction of a quantity of mucus which replaces a portion of the contained air. When sufficient mucus has reached the ostium by ciliary action to fill it completely so that incoming air cannot pass by, negative pressure develops in the sinus. The pressure falls as the mucus mass is progressively dragged and pushed through the ostium. When the effective power of the cilia is equalled by the difference between atmospheric pressure and the pressure within the sinus, the mass ceases to advance, and the pressure ceases to fall. The cilia continue to act, and remove the mucus in thin films. When sufficient mucus has been removed, atmospheric pressure forces air through into the sinus again.

Negative pressure almost certainly develops clinically in sinuses when they are more or less filled with mucus, and when their cilia are active.

The negative pressure, which is known to occur within the middle ear, is probably due to the force of ciliary action moving mucus down the eustachian tube.

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DISCUSSION

J. KENT LEASURE, M.D., Indianapolis, Ind.: Dr. Hilding has again established a corner around which medical thought must turn in the future. He has thrown a light upon conditions which in the past have been explained by gaseous absorption. His experiments are quite impressive and their simplicity makes them very conclusive. The more we learn about ciliated epithelium the more we are amazed with its action and significance.

As he has explained, anesthesia plays a big part in postoperative atelectasis. The frequent appearance of an allergic background in postoperative atelectasis must not be overlooked because here we have a condition which would also add to the amount of mucous production. Again since postoperative atelectasis occurs unilaterally so predominately one cannot help but wonder what selective factors determine which lobe or lung may collapse during a bilateral anesthesia.

As mentioned by Dr. Hilding, he presented a paper last year in which he described the effects of loss of ciliary function especially in asthma and influenza. At present, he has shown that ciliary action is responsible for the production of postoperative atelectasis. The thought then arises whether well recognized pre-operative asthma is present in cases where postoperative atelectasis occurs. In talking with several internists and looking over the literature, I find that as a rule the two do not appear together and atelectasis is not considered as a complication of asthma. Exceptions do occur, however, but rarely. Dr. Forry of the pathology department at Indiana University School of Medicine could furnish me with but one such postmortem report. In this case the operation was thoracic sympathectomy for the relief of asthma. The atelectasis of the left lung was "felt to the result of puncturing the pleura at operation, in spite of the fact that no puncture wound was demonstrated grossly, as the collapse was noted immediately following operation." Also in an article appearing in the Archives of Internal Medicine of 1930, Dr. Clarke refers to one case of postoperative atelectasis with a history of pre-operative asthma. These are rare exceptions to the rule and I think the rule still stands.

It would seem at first glance that an experiment on a chicken's trachea as described by the author would not apply to the ostium of a human sinus because of the shortness of some ostia. However, the length and toughness of the streams of mucous and purulent material which we see coming at times out of the ostia of sinuses would undoubtedly leave a partial vacuum behind even if it came out of the short sphenoid ostium.

In applying the experiments to the middle ear, I believe as Dr. Hilding does. In fact, during the discussion of a paper presented before the Indiana Academy of Ophthalmology and Otolaryngology in 1941 entitled "Tympanic Cavity Irrigation," I mentioned that perhaps the propulsive effect of the cilia of the eustachian tube beating in the proper direction may have more to do with recovery than the effect of any medication which might be used.

It has been a great pleasure to review Dr. Hilding's paper which displays his characteristic thoroughness.

A. J. CARLSON, Ph.D., Chicago, Ill. (by invitation — read in absentia).

1. Dr. Hilding has made a *new*, and in my judgment, *significant contribution* to the physiologic pathology of the lungs. The work should be extended on more experimental animals.
2. We should determine the time required for diffusion of air through a thin mucous diaphragm, when ciliary motion has ceased or become impotent, to equalize air pressure on both sides of the mucous diaphragm.
3. I agree with Dr. Hilding that the absorption of air into the blood of the occluded lung lobe unquestionably occurs. Also, we must not overlook the role that spasms of the bronchial muscles may play in the bronchial obstruction.

4. The mechanism or mechanisms producing the high negative pressure (up to 600 mm. H_2O) in cases of massive collapses of the lungs (assuming the findings are correct) are not accounted for by Dr. Hilding's present experiments, for adding any number of equally weak links into a chain does not make the chain stronger than its weakest link.

ALFRED LEWY, M.D., Chicago, Ill.: The characteristic, periodical pain in frontal sinus inflammation has always been more or less of a mystery. I don't know whether or not this explanation has been offered before, but Dr. Hilding's explanation seem to indicate that the mucus that probably starts downward when the patient first arises in the morning and assumes the upright position, through the combined action of the cilia and gravity, may produce sufficient vacuum in the frontal sinus to be responsible for the periodicity of the pain, that is, to bring it on at that period of the day when it is commonly noticed.

A. C. HILDING, M.D., Duluth, Minn.: I would like to thank the gentlemen for their discussions.

Unquestionably allergy may increase the production of mucus, and that may be one of the factors in the production of the mucus in atelectasis. There are many factors that could enter into such production. But whatever they are, there must be an excess of mucus before atelectasis develops. Why it happens in one lobe and on one side and not in another lobe on the other side seems to be owing to a combination of factors. The diameter of the tube, the excursion of the tube, and the changes in diameter and length, would enter into the problem as would the viscosity of the mucus, the amount of mucus, the position of the mucus with reference to gravity, and the force of respired air flow.

Atelectasis seems to be prevented and cured in most cases by simply turning the patient to a different position in bed and getting him to breathe deeply. Seemingly he need not breathe deeply continuously but only occasionally. This will upset the combination of circumstances that produces the mucous pistons.

It is true, as Dr. Carlson says, that adding any number of weak links to a chain does not increase the strength of the chain, but in a chain every link carries the whole load. The analogy does not seem to be quite pertinent in this discussion, however. In atelectasis there are a succession of disconnected pistons in a cylinder, each one with its own motive power, the high negative pressure reached being a summation of their individual effect. I believe that it is at least a possibility that this piston-cylinder mechanism of mucus and cilia is the explanation of even these great negative pressures. If this is not the case, then what is the force which holds the mass of slippery mucus suspended between atmospheric pressure and a pressure 60 cm. lower?

INTRINSIC ALLERGY AS IT AFFECTS THE EAR, NOSE AND THROAT: THE INTRINSIC ALLERGY SYNDROME

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IN ANY paper on allergy nowadays an explanation of what is to be included under the term should be attempted. Therefore I shall comment first on intrinsic or physical allergy. With this I shall mention briefly certain background subjects, such as desensitization, the alarm reaction and the known functions of the adrenal cortex before I describe the syndrome which I have chosen to call the "syndrome of intrinsic allergy." Before proceeding with this discussion, I wish to acknowledge the advice and help which Dr. C. F. Code gave me in this study.

INTRINSIC OR PHYSICAL ALLERGY

The first recognized allergic reaction, the tuberculin reaction, was of the antigen antibody type, and as pointed out by Kahn,[43] was a cellular rather than a humoral reaction. Kahn expresed the belief that allergic reactions of tissue are hyperimmune reactions in which the combination of antigen with antibody not yet released from the cell produces cellular disruption. This describes the so-called immunologic or antigen antibody type of allergy.

A great deal of work recently summarized by Rackemann,[68] Kellaway[44] and Farmer[24] suggests that many, if not all, of the phenomena which are recognized as allergic are produced by cellular injury with release of histamine which has been shown to be a normal constituent of most tissues. Histamine released into the tissue spaces acts on the arterioles and capillaries and produces arteriolar constriction and capillary dilatation in human beings, according to Wagener,[88] and also in animals, according to Dale and Laidlaw[17] and Best and McHenry.[9] In addition the permeability of the capillary walls increases and localized edema occurs subsequently.

Lewis[54] and his associates showed that in the skin of human beings sensitized by exposure to physical irritants, such as ultraviolet light and burning, further exposure to such irritants after a latent period produces a reaction indistinguishable from that produced by the injection of histamine. Kellgren[45] felt that the production of a painful region in muscles, known as myalgia, by exposure to physical stimuli was a similar phenomenon to the cutaneous reaction produced by physical agents.

Lewis, Grant and Marvin[55] showed that following stimulation of the skin of patients sensitized by physical agents, a condition of partial or complete refractoriness to further stimulation of the same kind was established. As this condition of refractoriness became established, the increased permeability of the capillaries of the skin disappeared.

Rose[70] stated that it was possible to distinguish between the immunologic or antigen antibody type of allergy, examples of which are asthma, hay fever and other conditions based on immunologic reactions, and a second type of allergy, namely physical or intrinsic allergy, which is not believed to depend on an immunologic reaction. Sensitivity to heat, cold and changes in atmospheric

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pressure,[56] endocrine dysfunctions,[25,42] and emotional disturbances[82,83,23] are examples of intrinsic or physical allergy. Code[13,14] has shown that in experimental anaphylaxis in animals histamine is liberated and Rose pointed out that the evidence on release of histamine in association with allergic phenomena, indirect though it is, has been found in patients who have had the second type of allergy. Evidence of a more direct nature has been obtained by Rose also. With the method devised by Code the histamine content of the blood was measured in a group of normal persons and was found to remain fairly constant. In a group of cases of intrinsic allergy Rose found the histamine content of the circulating blood may be increased after stimulation of the skin by exposure to the appropriate stimulus.

Hanhart[8] found in investigations of 70 large families in which there was a history of allergy that the general idiosyncratic disposition toward intrinsic allergy was inherited as a mendelian dominant. He stated that although severe hay fever with pollen asthma may occur in well-balanced persons, perennial vasomotor rhinitis with bronchial asthma (a representative of the intrinsic allergy group) nearly always is indicative of neuropathic or psychopathic personality and an unstable autonomic nervous system. He concluded that the hereditary disposition is far more important in the occurrence of the physical or intrinsic group of allergies than external influences, such as local sensitization through inflammation or trauma.

Berger[8] stated that a definite group of nonallergic causes consisting of physical, chemical and hormonal conditions and psychogenic reflexes is associated with allergic conditions. Any of these may produce any of the allergic conditions. The characteristic feature in such conditions is a commonly inherited or acquired constitution of certain tissues, particularly of the peripheral blood vessels, connective tissues and the nerve ends. These tissues have a definite susceptibility for selected and preferred stimuli and tend to respond in typical ways.

Muzio[63] has shown that the immediate response of nasal mucosa to changes in temperature affecting the skin is mediated through the automonic nervous system. Larsell and Fenton[52] have submitted evidence to suggest that intrinsic allergy, particularly as it affects the nose, produces the same effect as that which would be obtained by stimulating the vidian nerve after removal of the superior cervical ganglion and after sufficient time has elapsed for degeneration of the fibers from the cells in the superior cervical ganglion. They felt that the clinical picture of congestion of the nasal membranes, with watery discharge, associated with various disease conditions may have its physiologic explanation in the overflow of vasodilator impulses to the nasal mucous membranes. They suggested that when such overflow is accompanied by abundant nasal secretion, the overflow is produced by the parasympathetic or cranial autonomic fibers since the secretomotor fibers belong to this system.[53]

Grant, Pearson and Comeau[29] showed that urticaria could be provoked in certain persons by both psychogenic and physical influences. These authors concluded that H substance (histamine) was released from cells of the skin through the action of peripheral nerves belonging to the parasympathetic or cholinergic nerves. Grant and his co-workers felt that the release of acetylcholine in turn led to the liberation of histamine from the skin cells. They did not consider that their work indicated that the activity of the nerves involved was in any way abnormal. They felt rather that the skin of their patients responded abnormally to a normal release of acetylcho-

line at the nerve endings. They also found that portions of skin of sensitized persons in which a wheal had been raised by the action of nerves failed again to respond with a wheal until after an interval of about two days. They felt that they might say that the urticaria is due to the skin cells' having acquired a sensitivity to acetylcholine and that when the cells once react with it, this sensitivity is discharged and requires a period of about two days for its re-establishment. They furnished evidence that the unresponsiveness was to some extent specific for the stimulus provoking it.

This evidence in favor of the cholinergic origin of urticaria in sensitized persons may be compared with that submitted by Larsell and Fenton in regard to vasomotor rhinitis. It seems a reasonable assumption that if a similar mechanism can be found at the basis of two manifestations of physical allergy, that all manifestations of physical allergy are probably mediated by a similar mechanism, that is, the setting free of histamine from certain abnormal cells by the action of cholinergic nerves.

That there is some abnormality in the sympathico-adrenal mechanism is suggested by the observation that patients displaying the syndrome of physical allergy do not seem to react to stress in an efficient manner.

In discussing the autonomic nervous system Cannon[11] stated that the ways have been revealed in which pain, cold, various emotional states and other conditions which excite autonomic discharges can induce or participate in pathologic processes. Thereby morbid phenomena which have long been regarded as mysterious have been explained in a reasonable way. Cannon[12] stated that he and his co-workers were able to show that muscular work, emotional excitement, asphyxia, low blood pressure, external cold, infection and hypoglycemia were accompanied by an extra secretion from the adrenal medulla and extra activity of the sympathico-adrenal system. Such services performed by the sympathico-adrenal system at times of unusual or critical need, Cannon has termed its "emergency" functions. Cannon showed that experimental animals deprived of the sympathico-adrenal system were normal so long as they were not subjected to stress.

White and Smithwick[90] stated that according to the evidence available the parasympathetic system never reacts with a diffuse discharge to "natural" stimuli. This suggests that an abnormality may be present in the vasomotor nervous system of persons who have physical allergy.

Selye[74] has stated that whenever the organism is placed in a critical situation it meets it with a typical tissue defense reaction. Selye felt that at least some of the symptoms of the alarm reaction are due to the liberation of histamine from various tissues of the body. He concluded from his study that one of the functions of the adrenal glands is to counteract or detoxify this metabolite. Exposure to variable surrounding temperatures and excessive fatigue are among the agents likely to act as alarming stimuli.

Kendall[49] stated that the changes in the distribution and excretion of inorganic ions and in carbohydrate metabolism which occur in adrenal deficiency are associated with the metabolic processes which involve fats and proteins. It is, therefore, not surprising to find a subnormal state in many physiologic processes in association with deficiency of the adrenal gland. The physiologic processes likely to show this subnormal state are the maintenance of blood pressure and body temperature and resistance to stress, long continued muscular

activity and toxic substances. Kendall stated that the known physiologic processes which are affected by the hormones of the adrenal cortex can be grouped in three divisions. These are: (1) the control of permeability and the transfer of inorganic ions and water between extracellular and intracellular phases and from blood to urine; (2) the activity of tissues with specialized functions, such as liver, kidney, gastro-intestinal tract and muscle; after adrenalectomy these physiologic processes are disturbed in large part because of changes in permeability and in the distribution of water and inorganic ions, especially sodium and potassium; and (3) the activation of enzymes. As stated previously, increased permeability of the blood vessels is characteristic of all allergy whether of the physical or the antigen antibody type.

Sensitization. — Duke[21] was of the opinion that specific hypersensitivity to physical agents could be the result of the development of a chemical substance in certain tissue. Following such sensitization, stimuli from the vasomotor system will release the chemical substance or substances to produce either general or local reactions.

The work of Lewis[56] on sensitization of skin to physical irritants, such as heat and cold, suggested the manner in which blood vessels might become sensitized. He also noted that such sensitization took place more readily in the presence of infection.

Danielopolu and Marcou[18] found that in an adrenalectomized animal after all traces of vasoconstrictive action of epinephrine had disappeared, if only a trace of epinephrine was injected into the animal, the blood vessels immediately regained their initial tonus.

Mechanism. — The evidence in regard to intrinsic allergy which I have reviewed briefly is suggestive of a fundamental, inherited, localized, cellular abnormality resulting in some change in cellular permeability and concomitant release of histamine when the deficient cell is affected by stimuli which obtain little or no response from the normal cell. The disorganization produced as a result of the stimulus is an injury response of the cell causing release of histamine with subsequent vasodilatation and pain. There is evidence to show that the sympathetic and parasympathetic action may not be normal, in addition to carrying stimuli to an abnormal cellular substratum.

GENERAL DESCRIPTION OF TYPE OF CASE AND THE PROBLEM UNDER CONSIDERATION

I have considered intrinsic allergy at some length because, although well recognized by investigators, this concept of allergy has not yet received wide publicity and because in the practice of otology and rhinology the type of allergy we have to deal with is usually of the intrinsic type.

Allergies, affecting the nose, paranasal sinuses and the ear, have plagued the otorhinologist since the inception of the specialty because of the associated infection and pain in the head.

Patients who have vertigo or pain in the head frequently consult the otorhinologist because the pain followed severe infection of the upper respiratory tract, frank suppurative sinusitis, or otitis media and mastoiditis although signs of such infection may have completely disappeared. More frequently the pain in the head is associated with nasal discharge which the patient may term "cold in the head" or "sinusitis," or with a chronically draining ear which the patient may believe indicates involvement of the mastoid process with impending intracranial complications.

The diagnosis of pain in the head associated with acute inflammation of the

paranasal sinuses has never been the subject of controversy. The pain and tenderness are in the same place over one of the sinus walls; the pain is of the superficial type. The same may be said of acute mastoiditis. In chronic suppurative sinusitis and otitis pain in the head rarely occurs. The point most striking to me about uncomplicated suppurative otitis and sinusitis is that reference of the pain to a distance does not occur as far as I have observed.

In hyperplastic sinusitis which is a condition in which symptoms and signs of vasomotor rhinitis and suppuration are intermingled, the picture is radically different. In such cases referred pains and bizarre headaches are frequent. It is in regard to this type of case that the controversy over the description and cause of the headaches and other symptoms has arisen. It has arisen because the headache does not seem to have a close anatomic connection with the inflammatory condition in the nose or ear that is presumed to have caused it. The patients have difficulty in finding a simile to describe their subjective sensations. The pains tend to be referred. The distribution of pain seems impossible on the basis of the known distribution of the cutaneous sensory nerves.

Therapeutic relief by both surgical procedures and nerve blocking has been inconstant, usually futile. The patient and a series of physicians have become discouraged; the patient's condition has become worse. The majority of this group of patients make the self-diagnosis of "sinus headache." Since, as I have indicated previously, confusion does not arise in cases of frank suppuration, it seems apparent that the pain in the head is the result of allergy rather than of infection. In accord with this concept many persons can be found who do not have sinusitis or chronic aural suppuration but do have the bizarre distribution of pain in the head.

Until recently the idea of infection as the cause of headache has been so firmly implanted in rhinologists' minds, however, that whenever pain in the head was present and evidence of frank suppuration could not be found, the phenomenon was explained on the basis of nonsuppurative infection characterized by edema and polypoid formation. Vast numbers of patients who had hyperplastic sinusitis were subjected to operative procedures as a result of this hypothesis with such an obvious lack of success that the idea grew up among both physicians and the laity that sinusitis could not be cured by surgical procedures. The abolition of the false concept of nonsuppurative infection has permitted consideration of pain and vertigo just described for what they really are, manifestations of allergy, and in this way has cleared up much of the confusion in regard to them.

Unfortunately, treatment by routine allergic methods has failed to be of help in many instances. It was not until these cases were recognized as primarily instances of physical or intrinsic allergy that progress was made in relieving the patient of the symptoms for which he needed relief.

SLUDER'S SYNDROME

Sluder[78,79] (1908) was the first to recognize the essential unity of the group of symptoms under discussion, but unfortunately was one of the principal proponents of the nonsuppurative infection theory. For the purpose of comparing the Sluder syndrome with the syndrome of "intrinsic allergy" which I will describe later, I have summarized the symptoms and signs of Sluder's syndrome from his monograph[80] as follows:

I. Background

A. Often more or less severe inflammatory disease before development of the syndrome

B. Vasomotor rhinitis, asthma and myalgia often associated with the headache (or head pain)

II. General symptoms

A. Pain—unilateral or bilateral: occurrence in acute exacerbations or "attacks" on a substratum of discomfort; "sense of muscular tension"; not otherwise described
   1. At root of nose
   2. In and about eye
   3. In upper jaw and teeth
   4. Extended backward to the temple and along the zygoma to the ear
   5. Extending down the posterior aspect of mastoid to neck
   6. Reaching backward from the mastoid to the occiput
   7. Extending to the shoulder, less often to the axilla and breast
   8. Occasionally extending to the shoulder and arm
   9. Referred to the vertex

B. Tenderness
   1. Severest at a point 5 cm. back of the tip of mastoid
   2. Associated with stiffness in "the muscles of the neck" after an attack

C. Vasodilatation, flushing and sense of heat over face

III. Symptoms referable to the ear

A. Tinnitus—character not described
B. Tubotympanitis (stuffy ears)
C. Vertigo — usually mild but at times comparable to true Ménière's syndrome

IV. Symptoms referable to the nose

A. Stuffiness (localization to one side not described)
B. Watery discharge
C. Itching

V. Symptoms referable to the mouth and throat

A. Glossodynia or burning of tongue (common)
B. Parageusia (rare)
C. Salivation (rare)
D. Pain on swallowing
E. Globus
F. Itching of hard palate and tonsillar pillars

VI. Symptoms referable to the eye

A. Conjunctival injection
B. Lacrimation
C. Either dilatation or narrowing of pupil
D. Widening or narrowing of palpebral fissure
E. Asthenopia
F. Photophobia—occasionally a sense of photophobia when no light is present

VII. Treatment

A. Cocainization of sphenopalatine region in nose for temporary relief of symptoms
B. Operation on "involved sinuses" as advocated by Sluder for more permanent relief
   1. Frequent failure to give relief

While refinements and additions in observation and description have been made since Sluder's final monograph, the referred pains in the head and signs and symptoms associated with hyperplastic sinusitis and aural disease still appear to be those recognized by Sluder. One is amazed that in complete lack of knowledge of the physiologic and anatomic bases of these symptoms and signs he should have been able to perceive a fundamental unity.

SYNDROME OF INTRINSIC ALLERGY

For purposes of description I have termed an interrelated group of painful symptoms about the head "the syndrome of intrinsic allergy." To investigate the probable mechanism of the syndrome it can be broken down into four main divisions as Sluder's syndrome can be: namely into vasomotor rhinitis, Ménière's syndrome, myalgia and the vasodilating pain syndrome. These main divisions may occur singly or in combina-

tion. Even when the symptoms of one or another of these divisions are so prominent as to seem to form a unique syndrome, careful investigation sometimes will show that many symptoms or signs of the other three divisions are also present. Table 1, prepared after clinical observation of more than 300 patients, I believe, will reveal the essential clinical unity of these four main divisions.

Vasomotor rhinitis. — Lillie was one of the first rhinologists to insist that the treatment of rhinologic conditions should be based on physiology rather than anatomy. He recognized Sluder's syndrome as an entity but was dissatisfied with the results obtained by the surgical methods of treatment. He, therefore, asked Hansel, then a fellow in the Mayo Foundation, to study this question. As a result Hansel wrote and published a thesis on vasomotor rhinitis.[33] Allergy later became Hansel's major preoccupation and in many papers summarized in his monograph,[34] he was able to demonstrate the allergic nature of vasomotor rhinitis. By reviewing one hundred microscopic sections of material removed by Sluder in operations designed to relieve the Sluder syndrome he was able to show that what Sluder thought a peculiar type of infection characterized by polypoid hyperplasia and sphenopalatine ganglion neuralgia was really allergy.[35] In sixty-six of the one hundred microscopic sections reviewed there was preponderant eosinophilic infiltration; the other slides revealed either normal mucosa or acute infection. Hansel's work has established at least the vasomotor rhinitis component of the Sluder syndrome on an allergic basis.

In our present work at the Mayo Clinic we make the diagnosis of perennial vasomotor rhinitis on the basis of a history of paroxysmal attacks of sneezing followed by nasal congestion and watery discharge from the nose, nearly always bilateral, together with the observation of a pale edematous mucosa, especially over the turbinates, covered with a watery or mucoid discharge. If, in addition to these findings, an abundant purulent secretion bathes polyps, the condition is diagnosed as hyperplastic sinusitis which is considered in this paper as being perennial vasomotor rhinitis with secondary sinusitis.

Ménière's syndrome. — The diagnosis of Ménière's syndrome has been said to depend on sudden attacks of rotational dizziness or vertigo, tinnitus and deafness in the involved ear. Nystagmus is said to accompany the attack and to be rotatory or vertical with the quick component of the nystagmus either away from or toward the affected ear. There is no general agreement on this point, however. Nausea with cold sweat and vomiting is usually included. Most observers have thought that the condition is usually unilateral. Harris and Moore,[37] however, stated that bilateral Ménière's syndrome is relatively common.

The cause of Ménière's syndrome is unknown. Crowe[15] gave the following reasons among others for suspecting that the peripheral system of semicircular canals and cochlea rather than the nerve or vestibular nucleus was the seat of the disturbance. The vestibular symptoms of Ménière's syndrome differ only in intensity from those produced by the functional tests. The infrequency of vestibular symptoms from intracranial tumors argues against the disease being in the nerve or the central vestibular nucleus. The involvement of other cranial nerves in Ménière's syndrome is rare. Trauma of the vestibular nerve does not cause symptoms. Crowe was of the opinion that the strongest reasons for suspecting variable pressure and chemical changes in the membranous labyrinth as the cause of Ménière's syn-

drome were the fluctuating character of the deafness and the frequency and length of the remissions. Mygind and Dedering[64] pointed out that the symptoms could be controlled to some degree by dehydration. Furstenberg, Lashmet and Lathrop[28] stated that the balance between the sodium and potassium ions in the endolymph was concerned in the production of Ménière's syndrome and suggested a diet low in sodium ion supplemented with large doses of ammonium chloride. Talbott and Brown[84] felt that a high intake of potassium may be of more importance in controlling Ménière's syndrome than a low intake of sodium. In 4 cases studied during an attack of Ménière's syndrome an increased concentration of serum potassium and a decreased concentration of serum sodium were noted. These workers suggested the hypothesis that an increase in serum potassium indicates a loss of tissue potassium just as a high level of serum calcium accompanies a loss of calcium from the tissues. They suggested also that the depleted potassium in tissues can be restored and electrolyte equilibrium re-established by the administration of additional potassium.[85]

Hallpike and Cairns[31] were the first to report the pathologic changes in two authenticated cases of Ménière's syndrome. They reported gross dilatation of the endolymph system affecting chiefly the scala media of the cochlea and the saccule. Degenerative changes were present in Corti's organ. They reported a conspicuous absence of inflammatory change. Hallpike and Wright[32] reported similar observations in another case. These pathologic findings have been confirmed by Lindsay[59] and others. These findings are in accord with the concept of an allergic etiology for Ménière's syndrome. Lindsay felt that the roles of allergy and vasomotor instability played a part in the production of symptoms, although he stated that clinically there were strong contraindications to Ménière's syndrome being on an allergic basis, that allergic manifestations were rare in Ménière's syndrome and that attacks of vertigo were not common in allergy. Brown,[10] as evidence in opposition to Lindsay's opinion, found a history of "vasomotor imbalance" common in the family histories of patients suffering from Ménière's syndrome. Halle[38] pointed out the association of deafness, vertigo and nystagmus with myalgia as far back as 1911.

Shambaugh and Roberts[76] found that the detection of the allergic factor in Ménière's syndrome was most difficult owing to the unreliability of skin tests in this condition.

Atkinson[3] cited 4 cases of Ménière's syndrome reported by Dohlman[20] in which the etiologic factor was allergic and presented 2 cases of his own in which similar symptoms were relieved by elimination diets.

From my own observations, I do not believe that Ménière's syndrome can be sharply divided from vertigoes without deafness and tinnitus as suggested by Atkinson. In my cases I see a gradual gradation of postural vertigo, that is, vertigo brought on by sudden change of position, up through tinnitus and vertigo without loss of hearing to the so-called true Ménière's syndrome. Many patients exhibiting the diagnostic triad of vertigo, tinnitus and deafness also will have much more frequently minor episodes on sudden change of position which are typical of so-called postural vertigo rather than of Ménière's syndrome. For the purpose of this paper, however, a diagnosis of Ménière's syndrome has rested on the presence of vertigo, tinnitus and loss of hearing in the affected ear or ears.

I believe sufficient evidence has been produced for the tentative classification of Ménière's syndrome as an allergy and as belonging to the larger syndrome of

physical or intrinsic allergy as it involves the various structures of the head.

Myalgia. — Localized painful spots in muscles producing reference of pain in a distribution not that of the cutaneous nerves were first described by Froriep[27] in 1843. I have considered myalgia of the head and pharynx in other papers.[91,92] One of the earliest and most enlightened discussions on myalgia was held in the Senate Chamber of the State Capitol of Minnesota in St. Paul on June 19, 1877, by the Committee on the Practice of Medicine of the Minnesota State Medical Society.[26] In the course of the discussion it was mentioned that the tissue changes produced by the "rheumatic agency" were not observed in myalgia or "muscle rheumatism." The tissue changes in "rheumatism" had been pointed out by Virchow and Froriep[87] to be hyperemia, serous exudations, inflammatory proliferation of connective tissue and changes in the sarcolemma and neurilemma. These changes were found to be absent in myalgia. In the discussion by the Committee on the Practice of Medicine just mentioned myalgia was thought to be a metabolic disturbance in muscle, which possibly depended on the lack of elimination of lactic acid and produced an irritation of the nerve endings in the muscle. It was thought that this metabolic disturbance depended on the premature checking of blood flow to the muscle and as a consequence the requisite nutrient supplies were withheld, the interchanges that reduced irritant to inert products were impeded, and the irritant bodies were detained within the muscle. It also was stated that atmospheric pressure had a decided effect in precipitating myalgia and the effect of drafts and sudden cooling in bringing on attacks was pointed out. It was observed that the Chippewa Indians, especially the squaws, were very likely to have myalgia. For treatment, opium and its derivates and salts of potassium were recommended. The local measures advised were heat and massage. At this meeting too it was mentioned that salicylic acid and salicin, drugs that seem to be almost specific in the treatment of fibrositis, had no influence whatever on myalgia. Few of the later papers on myalgia have exhibited the knowledge of physiologic possibilities presented in this early discussion.

Sluder[81] stated that cases reported as myalgia in the literature were in fact instances of his syndrome.

Lange[50,51] described myalgia under the title of "myogelosis." He found that macroscopic and microscopic examinations of tissue excised from the tender region did not reveal pathologic change. A number of observations demonstrated that myogelosis does not disappear during deep general anesthesia. The absence of pathologic change is consistent with an allergic etiology of myalgia. Lange concluded that myogelosis is not a circumscribed spasm of muscle fibers but is the result of an organic change probably colloid chemical in nature. Maliwa[60] presented evidence that myalgia occurred in combination with allergies. Sell[73] stated that muscles with a chiefly postural function contain more sarcoplasm than muscles for quick motion, and that those rich in sarcoplasm are the ones predisposed to myogelosis.

It remained for Lewis[56] and Kellgren[46,47,48] and associates, however, to establish myalgia on an experimental basis. They demonstrated that the pain in myalgia is the same as that produced by exercising a muscle under ischemic conditions and that in myalgia the pain from a given muscle is felt over definite regions and seems to follow a segmental pattern. Lewis concluded that the pain from a given muscle is determined by a chemical or physiochemical stimulus developed in the muscle mass, which he called the factor "P" and that the process

was rapidly reversible in the presence of oxygenated blood. Lewis found the pain from muscle to be "deep pain" which is not well localized, because impulses from visceral and parietal structures are recognized as pain and referred to the whole muscle segment rather than accurately localized. He stated that sharper localization than this had not been found useful in phylogenetic development. Lewis also has shown that skin may be sensitized to physical agents so that a further exposure to such agents will produce a reaction characterized by release of histamine. In this he anticipated the recent observations on immersion foot. Lewis was of the opinion that the process in muscle producing myalgia was similar to that which he had observed in the sensitization of skin.

In observing the mechanism of pain in the head produced by histamine, Pickering and Hess[65] suggested that the headache produced by the intravenous injection of histamine would be explained completely if the headache were due to stretching of a sensitive structure lying close to the meningeal arteries. Such stretching might result from swelling of the perivascular tissues or from widening of the meningeal arteries. They showed that while amyl nitrite will produce an expansion of the brain and a lowering of the blood pressure identical to that produced by histamine, it will not produce headache. It is known that histamine increases the permeability of the minute blood vessels which amyl nitrite has not been shown to do. An increase in vascular permeability might be a factor in producing headache if the pain were due to stretching of the perivascular structure from edema secondary to the increased permeability of the capillaries.

The studies of Lewis[56] and his coworkers lead to the hypothesis that in myalgia due to local sensitization of cells, local arteriolar vasoconstriction might lead to a local deficit in oxygen so that normal reversal of the catabolic process could not take place. Such failure would result in the piling up of enough of the P substance to produce pain. That certain vasodilating substances also are produced, possibly histamine, is suggested by the fact that in the exacerbation of severe pain called "attacks" the pain at least at first is pulsating. In the group of cases considered in this paper the diagnosis of myalgia of the head or pharynx has been based on precipitation of a painful syndrome in the neck and head by sudden changes in temperature or barometric pressure. The pain was associated usually with unilateral tenderness and stiffness in certain groups of muscles, which had none of the characteristics of fibrositis (jelling, relief with mild exercise, relief from salicylates and tendency to recur in different locations) and with pain referred to the regions worked out by Cyriax.[16] The differential diagnosis from migraine and other conditions with which myalgia might be confused is covered in previous papers on myalgia of the head and myalgia of the pharynx.

Vasodilating pain syndrome.—Wolff[93] demonstrated that traction on, or dilatation of, the cranial arteries will produce pain but crushing or spasm will not.

Schumacher, Ray and Wolff[71] stated that the large arteries at the base of the brain, which include the internal carotid, the vertebral, the basilar artery and the proximal portions of their branches, are the primary sites of origin of pain in the head resulting from histamine, although the extracranial arteries are subsidiary sources of pain.

The work of Pool and Nason[66] suggests that the branches of the external carotid artery are adrenergic and the branches of the internal carotid artery are cholinergic. Thus epinephrine given

intravenously, while relieving the pain of the vasodilating pain syndrome involving the extracranial vessels, would tend to accentuate the pain originating from intracranial vessels as in migraine.

Ray and Wolff[71,69] described six basic mechanisms of headache chief of which were dilatation and traction on the venous sinuses and intracranial and extracranial arteries. They stated that headache from intracranial disease is usually referred pain. They also found that the chief pathway for sensation of pain in the head was by way of the fifth cranial nerve but that the seventh and ninth cranial nerves and the first three cervical nerves also served as pathways.

Horton, MacLean and Craig[38] showed that a vasodilating headache may be produced by injections of histamine insufficient to provoke a histamine headache; the pain starts from thirty minutes to an hour after the injection of histamine. This suggests some intermediate mechanism by which the injection of histamine sets free, as Pickering and Hess suggested, after a latent period, the P substance of Lewis, possibly histamine, at the same time producing vasodilatation.

That parasympathetic overaction is the principal factor in both migraine and the vasodilating pain syndrome is indicated by the findings of Schumacher, Ray and Wolff[71] and of Horton, MacLean and Craig[39] that cervical sympathectomy has no effect in preventing the development of either migraine or vasodilating pain syndrome. Horton[40] also demonstrated that increased sweating, except for the circumoral sweat glands, a cholinergic phenomenon, accompanies the vasodilating pain syndrome. Schumacher and Wolff[72] showed that in migraine central vasoconstriction produces the prodromal scotomata. This might represent a preliminary action of the sympathetic nervous system followed by a powerful parasympathetic reaction. This suggests a mechanism, vasoconstriction followed by dilatation, by which not only the vasodilating pain syndrome but all the phenomena under consideration might be precipitated.

I have found that watery discharge from the nose in vasomotor rhinitis and the head pains of myalgia can be activated after a latent period, similar to that reported by Horton, MacLean and Craig in cases of histaminic cephalgia, by injections of histamine subcutaneously. That the effects of subcutaneous and intravenous injections of histamine are comparable is suggested by the studies of Kahn[1] on the rate of diffusion of histamine injected subcutaneously. I feel, therefore, that the difference between the histaminic cephalgia of Horton and the exacerbations of headache seen in myalgia, vasomotor rhinitis and hyperplastic sinusitis and Ménière's syndrome are differences in degree rather than in kind. This idea is further buttressed by the finding of Wolff, Hardy and Goodell[94] that if a painful stimulus produced by the concentrated thermal radiation from a 1,000 watt lamp was allowed to fall on the forehead during a histaminic headache, it was possible by increasing the intensity of the secondarily induced pain to reach a level that made the underlying headache imperceptible. These authors also found that pain sense had no spatial summation. Expressed more simply, this hypothesis is that the total effect of several intensities of pain acting simultaneously is equal not to their sum but merely to the intensity of the severest pain. It can be seen that an intense pain from one part of a stimulated vessel would overshadow the appreciation of pain stimuli from other parts. It can be observed in cases of conjoined myalgia and vasodilating head pains that the more severe the vasodilating headache becomes the less appreciation the patient has for subsidiary pains which are only noticeable when the pain is in-

creasing or diminishing or if the vasodilating factor of the pain does not reach an intensity sufficient to abolish the appreciation of less severe pains.

Horton has demonstrated that histaminic cephalgia probably involves branches of the external carotid artery. It has been shown that ergotamine tartrate and epinephrine contract the extracranial vessels and these drugs will relieve the pain of histaminic cephalgia.[66] That the extracranial arteries are a source of pain in myalgia also is suggested by the fact that intravenous injection of epinephrine will relieve the pain. Relief of pain on injection of epinephrine also indicates a similarity between the vasodilating headache of the histaminic cephalgia type and the headache associated with the myalgia and distinguishes both from migraine, because in the first two types of headaches the extracranial arteries are chiefly involved, while in the latter the intracranial vessels also may be a source of pain.[72]

In the cases considered as vasodilating pain in this paper the diagnosis rested on the presence of "visceral" pain in the head, sudden in onset, of a pulsating character unless extremely severe, usually unilateral but occasionally bilateral or midline. When the pain occurred in the anterior portion of the head, it usually was associated with lacrimation, conjunctival injection, swelling of the eyelids, congestion and discharge on the same side as the pain or on both sides if the pain was bilateral. The pain tended to appear frequently at night, would awaken the patient and was usually less severe if the patient stood upright than if he lay down. When the pain was present and often in intervals free of pain, the common carotid and its branches and the occipital artery were tender to pressure. Occlusion of the vessels tended to relieve the pain which flooded back when the pressure on the vessels was released. This type of pain in the head or headache has not been observed in any case in this series in which the patient was less than thirty years of age.

Diagnosis in 362 cases drawn from the files. — Three hundred sixty-two case records drawn from the files were studied. In each case the painful symptoms about the head which I have now included in the syndrome of intrinsic allergy were present. The clinical diagnosis in this group of cases is given in table 2.

In 173 cases a diagnosis of perennial vasomotor rhinitis only was made and in 77 vasomotor rhinitis was found to be associated with one or the other of the division of symptoms previously mentioned. I believe the grouping in table 2 bears such a similarity to the syndrome proposed by Sluder that the syndrome of physical or intrinsic allergy and Sluder's syndrome may be considered identical.

On reviewing the records of the patients studied in preparing this report, I noted that patients who had perennial vasomotor rhinitis almost invariably stated that sudden change of temperature affecting the surface of the skin was a precipitating factor in their complaint. In myalgia of the head and pharynx a history of the appearance of the complaint following fever or exposure to extreme cold was often obtained. In myalgia a history of exacerbation of symptoms following sudden changes of barometric pressure, such as in approaching storms, as well as the precipitating effect of sudden chilling of the skin was obtained. In the vasodilating pain syndrome and Ménière's syndrome, I have rarely been able to obtain a history of precipitation by external physical agents except when vasomotor rhinitis or myalgia was associated.

It is this definite, fairly frequent association, however, which has led me to include the vasodilating pain syndrome

and Ménière's syndrome in the syndrome of physical allergy.

REVIEW OF TYPES OF TREATMENT EMPLOYED FOR MEMBERS OF THE SYNDROME OF INTRINSIC ALLERGY IN GENERAL

The first treatment suggested for a member of this symptom complex was that of Sluder in 1908 who anesthetized the sphenopalatine ganglion to relieve neuralgia of the sphenopalatine ganglion. He later suggested injection of the sphenopalatine ganglion with phenol. Lillie and Childrey[58] have shown that cauterization of the filaments emerging from the sphenopalatine ganglion will destroy them and, if the ganglion is superficially placed, it actually will destroy the sphenopalatine ganglion itself. They reported relief of symptoms after application of silver nitrate. Sewall[75] suggested actual removal of the sphenopalatine ganglion.

In a recent review of neuralgia of the sphenopalatine ganglion Eagle[22] stated that the condition is characterized by unilateral pain in the face which never extends above the level of the ear. The pain is lancinating and continuous. Usually there are two main points of maximal pain, one in the region of the orbit and the other just posterior to the mastoid process of the temporal bone. Eagle mentioned reference of pain, the occurrence of vasomotor changes on the homolateral side of the nose and timnitus on the homolateral side with vertigo. Eagle expressed the belief that nasal contact was the usual cause of sphenopalatine neuralgia and found that 80 per cent of 159 patients required some type of intranasal correction. He stated that all except 1 of the 53 patients operated on were relieved of their symptoms. Martin,[61] discussing the same syndrome, stated that the pain was transmitted along the arterial tree, through the carotid sheath to the cervical and thoracic portions of the spinal cord. He reported a case in which temporary relief was obtained by resection of the sphenopalatine ganglion and concluded that resection of the sphenopalatine ganglion did not give permanent relief to neuralgia of the sphenopalatine ganglion because the efferent and afferent nerves of the periarterial plexus took over the function of the ablated nerves. This was indicated by the return of the vasomotor response.

In the cases I have studied I have found that cocainization of the sphenopalatine ganglion will relieve temporarily many of the pains that occur about the face and forehead, but that it usually does not relieve pain in the temples and cranial regions.

A few cases have been found in which submucous resection was necessary to get rid of a "trigger" area in the nose, but in these subsequent treatment with nicotinic acid or histamine was required to relieve the pain. At the Mayo Clinic we have not been able to equal Eagle's record in relieving sphenopalatine neuralgia by submucous resection alone, but we can confirm his statement that in certain instances such a result has been secured.

Various types of treatment have been suggested for Ménière's syndrome. Although most of these were mentioned in connection with the description of Ménière's syndrome, I shall list them briefly here: administration of potassium to control electrolyte equilibrium; the Furstenberg regimen and general measures including elimination of foci of infection.

Simonton[77] noted relief from vertigo by the Furstenberg regimen in 34 per cent of cases, partial relief in 28 per cent and no relief in 38 per cent of the cases studied. Walsh and Adson[89] found that 36 per cent of the patients in their series were relieved of vertigo on the Furstenberg regimen, 26 per cent were improved and about 39 per cent showed

no improvement. They found that on administration of potassium nitrate vertigo disappeared in 31 per cent of their cases, improvement occurred in 46 per cent and in 23 per cent no improvement occurred. Of 22 patients treated by other general measures, including elimination of foci of infection, relief or improvement was obtained by 78 per cent, but in this group the improvement did not immediately follow treatment. The statistics of Simonton and of Walsh and Adson in regard to medical treatment are based on a period of observation of from six to eight months. The results of Walsh and Adson indicated that "general measures with removal of foci" gave better eventual results than medical treatment. This unusual result probably depends on the tendency toward spontaneous remission in Ménière's syndrome.

House dust. — Hansel[36] found that in treating allergies of the nose with house dust that satisfactory results were dependent on optimal dosage. He observed that poor results usually were due to starting with too strong a dose or overstepping tolerance with a dosage which was too high. He was of the opinion that dust extracts contain some specific unknown antigen. He, like Shambaugh, was dissatisfied with the usual allergic handling in many of these cases except with house dust because of the questionable results from skin testing. In the light of the work of Selye in regard to the alarm reaction it would seem possible that results of treatment with house dust depend on the irritant action of the dust rather than on the action of an unknown antigen.

Since therapeutic dosages of dust were of the same order as those found effective in histamine therapy, it was thought the "unknown antigen" might be histamine. Although Code has shown that dust extracts do not contain histamine, I have found that general principles outlined by Hansel as applying to dust therapy also apply to histamine and nicotinic acid therapy.

Histamine. — In 1929, Horton and Brown[41] suggested that since the patient who is sensitive to cold is also sensitive to a chemical substance formed in the tissues of the body, autodesensitization would be possible. Two procedures were considered: (1) desensitization by repeated injections of histamine over long periods in doses so minute that they would be insufficient to produce a systemic effect and gradual increase in the doses, and (2) autodesensitization by daily immersion of the hands in cold water of decreasing temperature for increasing periods. At that time Horton and Brown chose the second procedure.

The first use of histamine in the group of cases of intrinsic allergy of the ear, nose and throat was apparently by Deutsch, who treated myalgia with histamine iontophoresis; he reported his results in 1931.[19] He based this treatment on the work of Lewis, von Trendelenberg, Ebblicke and Török who suggested that anoxemia secondary to vascular spasm was responsible for the accumulation of a product of metabolism which produced the local symptoms. Deutsch, therefore, introduced histamine into the involved muscles with favorable clinical results.

In 1939, Horton, MacLean and Craig[38] reported the use of histamine injection for patients with vasodilating pain syndromes in the head. This suggestion of Horton and Brown's[41] which has been carried out in many conditions since 1929 opened up the first hopeful new line of treatment of the conditions under consideration that had been discovered for many years.

In Alexander's[1] experience neither histamine nor histaminase had any value

in the treatment of extrinsic allergy in which the cause was a specific hypersensitiveness, but in other conditions, such as urticaria, or especially, intrinsic allergy, histaminase may be helpful.

Nicotinic acid. — The use of nicotinic acid in the syndrome under consideration was first suggested by Harris and Moore[37] for Ménière's syndrome in 1940. They adopted its use because a large percentage of pellagrins have rotational vertigo. They had tried thiamine chloride first without benefit. They then added nicotinic acid, 250 mg. daily, with entire relief of the symptoms of 17 out of 20 patients treated. This observation awakened considerable interest in the use of this drug in Ménière's syndrome and was followed by several papers by Atkinson[4,5,6] who used essentially the same treatment.

Aring and his co-workers[2] showed that nicotinic acid and quinine nicotinate administered intravenously increased the intracranial blood flow for periods lasting from twenty to sixty minutes. This effect paralleled the flushing of the skin. Nicotinic acid amide was found to have no significant effect on the total intracranial blood flow.

Unna[86] found the toxic dose of nicotinic acid in mice and rats to be 4 to 5 Gm. per kilogram of body weight following subcutaneous administration and 5 to 7 Gm. per kilogram following oral administration. The prolonged oral administration of 2 Gm. a day of nicotinate of sodium per kilogram of body weight to rats, chickens and dogs during a two months' period did not produce any symptoms of toxemia or pathologic alterations in any organ. Moore[62] gave nicotinic acid in doses of 250 grains (16 Gm.) a day to patients suffering from multiple sclerosis for periods up to a year without ill effect.

TRIAL OF THE SKIN TEST WITH HISTAMINE AS AN AID IN SELECTING TREATMENT IN CASES OF THE PHYSICAL ALLERGY SYNDROME

Atkinson felt that since there was evidence to show that the manifestations of allergy were due to the release of histamine, or a histamine-like substance, that intradermal testing with histamine could divide patients who had Ménière's syndrome into a group that was sensitive to histamine and another larger group that was insensitive to histamine. He cited the previous report of Dzsynich and Gallé as to the use of histamine as a general indicator of allergy. Dzsynich and Gallé injected 0.001 and 0.00001 mg. of histamine in 0.1 cc. of saline solution beneath the skin of the forearm and reported that the test was positive for allergy when a wheal of 1 cm. and a surrounding flare of 2 cm. appeared. In his original paper Atkinson stated that he injected 0.01 mg. of histamine intradermally (which I assumed to be histamine base). In a second paper published in 1942 Atkinson[7] apparently continued to follow this same technic as his sole statement as to method was that the technic previously described should be followed closely to secure good results. In an article published in 1943[6] he stated that an intradermal test is performed by injection of 0.0057 mg. of histamine base (0.01 mg. of histamine dihydrochloride). If this latter dose is taken as the one used by Atkinson, it can be seen that the dosage used in the cases on which this paper is based contained 0.0043 mg. more histamine base than that proposed by Atkinson in his third paper but it is assumed that this variation could have little effect on the result. (The work reported in this paper was done before the correction in Atkinson's third paper was obtained.)

Atkinson apparently disregarded the area of erythema surrounding the wheal and based his conclusions as to sensitivi-

ty to histamine on the presence or absence of "pseudopods" from the wheal. Lewis and his colleagues[57] described the skin reaction to histamine as a "triple response" consisting of vasodilatation of the small arteries at the site of the injection, a diffuse flare and an area of local edema or wheal which appeared over the small area exhibiting vasodilatation. They showed that the vasodilatation and wheal are due to direct action on the capillary wall. The flare, however, is caused by a local axon nerve reflex. Local vasodilatation and wheal formation depend on the direct stimulation of the cells of the vessel walls by the irritating agent. The flare occurs only in sensitized skin and is thought to be a specific response to histamine. Atkinson paid but little attention to this in his test, however, and depended on the production of pseudopods which Lewis has shown to be due to the distribution of the local lymphatics.

Before proceeding with the treatment of patients with either histamine or nicotinic acid it was thought suitable to test the validity of Atkinson's hypothesis in cases without physical allergy and the whole group of cases of physical allergy syndrome.

Cases without physical allergy. — Fifteen patients who gave no history of physical allergy were subjected to the intracutaneous injection of histamine diphosphate. The dose was 0.01 cc. of a 1:1,000 dilution of histamine base. Negative results (no pseudopod formation) were obtained in 8 cases; positive results (one or more pseudopods extending at least 1 cm. from the edge of the wheal) were obtained in 7 cases. One patient who had a strongly positive response to Atkinson's test with two large pseudopods, extending 4 cm. from the edge of the wheal, and three medium sized pseudopods, extending 2 cm. from the edge of the wheal, was found to have a brain tumor. Another was found to have a duodenal ulcer on roentgenologic examination. The general physical examinations of the other 5 patients gave essentially negative results.

Cases of physical allergy. — Fifty-five patients whose conditions had been diagnosed as the physical allergy syndrome were then subjected to the Atkinson test and subsequently treated with either histamine or nicotinic acid without regard to the results shown on test. The results of the test and of treatment are given in table 3. The result of treatment was considered good when complete or nearly complete relief of symptoms was maintained for three months or more and fair when only 50 to 90 per cent relief was maintained for three months or more.

In a further trial of whether skin tests with histamine were an aid in selecting treatment, 55 patients whose condition had been diagnosed as physical allergy were subjected to intracutaneous tests with histamine diphosphate; for these 0.02 cc. of a 1:50,000 dilution of histamine base was employed (table 4). The criterion for a positive test was a flare extending more than 1 cm. beyond the edge of the wheal in all directions. The tests were all made in a closed room at a temperature between 75 and 80 F. The criteria as to results of treatment were the same as in the previous tests (table 4).

Comment. — The following conclusions were drawn from these tests: 1. There does not seem to be any agreement between the results of skin testing with histamine and the results of treatment. 2. Skin tests with histamine seem valueless both for diagnosis and as a guide to therapy. Therefore, no further skin testing with histamine was done. 3. These findings do not bear out Atkinson's contention that vasospastic and vasodilating types of allergy exist, since

the response in cases of all types in the physical allergy syndrome was equally good to treatment with either histamine or nicotinic acid.

TREATMENT OF MY SERIES OF CASES

The cases of the syndrome of intrinsic allergy about which I am reporting the results of treatment do not correspond to the 362 cases which were drawn from the files for a study of diagnosis and symptoms when I was formulating my impressions about the interrelated group of symptoms which I have now termed "the syndrome of intrinsic allergy." The treatment series is in general a later group of cases although there may be some overlapping. The treatment series consists of 327 cases in which nicotinic acid or histamine or both were employed. Thirty-two cases in which nicotinic acid amide, the extract of whole adrenal cortex or epinephrine was given and 15 cases in which Kendall's extract of whole adrenal cortex and epinephrine were used are considered in addition.

Method of treatment with histamine. —The solution of histamine diphosphate used was equivalent to a 1:50,000 dilution of histamine base. After trial with various dosages, it was found that 0.1 cc. of this solution, if given subcutaneously, would not precipitate symptoms in the majority of cases of the physical or intrinsic allergy syndrome. In the few cases in which exacerbation of symptoms was likely to be produced by this dosage, injections were started with 0.1 cc. of 1:100,000 dilution of histamine base or if necessary 0.1 cc. of 1:200,000 dilution. No patients were found who had an untoward reaction to the dilution last mentioned.

Following the initial dose patients received subcutaneous injections twice daily; the quantity injected whatever the dilution selected, was increased each time by 0.1 cc. until symptoms disappeared or an objective return toward normal was observed in the appearance of the nasal mucosa in cases of perennial vasomotor rhinitis. The dosage which would maintain the patient symptom free was termed the "optimal dosage" following the nomenclature established by Hansel in the treatment of vasomotor rhinitis with dust extracts. It was found that a small increase of the dose beyond that producing the initial symptomatic relief would result in a reappearance of symptoms and each further increase would produce marked increase in the severity of the symptoms.

When the optimal dosage of the twice daily injections was established, half the total daily dose was given once daily to see if improvement was maintained. It often was found necessary to increase the dose slightly to continue the patient symptom free. This daily dose was termed "the maintenance dose" and was found to vary between 0.1 cc. of a 1:50,000 dilution of histamine base and 0.1 cc. of a 1:1,000 dilution of histamine base. The majority of patients, however, were maintained on 0.5 cc. of a 1:10,000 dilution of histamine base. An attempt was made in many cases to reduce the number of injections to either three or four weekly but such reduction was usually followed by recurrence of symptoms in a short time. The patients were advised, therefore, to continue the maintenance dose for six months and then discontinue treatment. In most instances in which this schedule was carried out, the original symptoms reappeared after a short time and either resumption of the daily injection of histamine or change to nicotinic acid by mouth was found necessary.

Method of treatment with nicotinic acid. — It was found that most patients could tolerate an initial dose of 25 mg. of nicotinic acid (niacin) subcutaneously. In a few instances this dosage produced a marked increase in symptoms

after a delay of from fifteen minutes to half an hour. In several cases in addition to generalized flushing of the skin, abdominal cramps and involuntary movement of the bowels occurred. In a few cases treatment had to be started with doses as low as 1 mg. of nicotinic acid given subcutaneously. In the average case, however, twice daily injections could be given, with an increase of 25 mg. on each injection until a dose of 100 mg. was reached. In the average case 100 mg. of nicotinic acid injected once daily was found to be an adequate maintenance dose. In a few instances the optimal dose was found to be as low as 12.5 mg. daily. An increase above this level resulted in reappearance of symptoms. In several other instances notably when vasodilating pain of the head was the chief presenting symptom, dosage of as much as 200 mg. daily had to be maintained. The cases in which deviation from a daily dose of 100 mg. was necessary were relatively few. For this reason, therefore, nicotinic acid is a more useful therapeutic agent than histamine diphosphate.

Patients were advised to remain on a daily dose of 100 mg. of nicotinic acid subcutaneously (when this was the optimal and maintenance dose) for three months. At the termination of this period they were advised to take by mouth 100 mg. of nicotinic acid night and morning as it was found that nicotinic acid by mouth was less effective in maintaining symptomatic relief than when it was administered subcutaneously. Patients were instructed to continue oral administration for three months and then attempt to discontinue treatment. In a few instances symptoms have not reappeared after discontinuing treatment for periods of three months but in most cases oral treatment has had to be continued. (I wish to express thanks to the Abbott Company for their kindness in furnishing a supply of nicotinic acid [nicamin Abbott] for use in this study.)

Results of treatment with histamine or nicotinic acid or both. — Results of treatment in 327 cases are summarized in table 5. Criteria for good and fair results and failure of treatment were the same as those employed in the trial of treatment in association with the cutaneous test with histamine. Various comments will be made also on details of the findings.

Perennial vasomotor rhinitis (table 5).—In 1 case in which a good result was obtained in the treatment of the vasomotor rhinitis with histamine and in another with nicotinic acid the associated asthma was not affected. In 3 of the 18 cases in which treatment with histamine was considered a failure, relief was obtained for two weeks, in 1 case for one week and in 1 for six weeks; in 1 case exacerbation of asthma was responsible for failure of treatment. In 1 case in which fair results were obtained from treatment with nicotinic acid septal deflection probably accounted for lack of relief.

Various cases in which treatment with nicotinic acid was classed as a failure should be mentioned briefly. In 2 of these cases in which immunologic allergy was associated, symptoms became severe when preseasonal "desensitization" was attempted after two months' relief. In 1 case of chronic ulcerative colitis, nicotinic acid semed to increase gastro-intestinal symptoms. In 2 cases asthma was precipitated by injection of as little as 1 mg. of niacin. In 1 case after two weeks' relief, a violent attack of vasomotor rhinitis was precipitated half an hour after injection (after initial flush of skin had disappeared). In 2 cases of immunologic allergy and positive skin tests relief was complete for one month. In 3 cases asthma developed after one month. The asthma, however, disappeared on cessation of treatment.

When both nicotinic acid and histamine were employed, success was obtained with nicotinic acid after failure with histamine in 3 cases and with histamine after failure with nicotinic acid in 1 case; 50 per cent relief was obtained with histamine after failure with nicotinic acid and histamine separately but complete relief on use of both together in 2 cases. Complete failure resulted from treatment with nicotinic acid and then with histamine in 6 cases. Simultaneous treatment with both was not tried in these cases. In 1 case failure resulted from histamine and an apparent good result was obtained for one month from niacin. Treatment in this case was terminated because of development of dermatitis of the hands. The dermatitis disappeared after treatment was stopped.

In another case an apparent good result with histamine disappeared after two weeks. With niacin the patient was relieved for two months before treatment had to be stopped because of the development of asthma, which disappeared on termination of treatment.

Perennial vasomotor rhinitis and Ménière's syndrome with or without myalgia of the head (table 5).—One of the patients who had symptoms of both perennial vasomotor rhinitis and Ménière's syndrome was given histamine both intravenously and subcutaneously without relief. Relief was obtained subsequently with niacin.

One of the patients who had vasomotor rhinitis, Ménière's syndrome and myalgia was completely relieved of all symptoms on subcutaneous treatment with histamine except at menses when a major attack lasting several hours would occur. Treatment with histamine, therefore, was classed as a failure. The patient subsequently was relieved completely by niacin.

Ménière's syndrome only or associated with exacerbation of myalgia (table 5).—One of the patients who had Ménière's syndrome associated with exacerbation of myalgia obtained relief of symptoms for two months on treatment with histamine, then had recurrence. This patient was subsequently relieved by nicotinic acid. Another patient who was having daily symptoms obtained complete relief on treatment with nicotinic acid for one month before recurrence.

Fair results (50 per cent relief of symptoms for three months or more) were obtained in 3 cases of Ménière's syndrome only on treatment with nicotinic acid. After two months' relief from 100 mg. of nicotinic acid daily, vertigo with abdominal cramps recurred in 1 case. This patient was completely relieved by reducing the dose of nicotinic acid to 50 mg. daily and supplementing it with 10 grains (0.65 Gm.) of potassium iodide with meals. In the 1 case in which failure from treatment with nicotinic acid occurred the patient first improved definitely for one month.

Vasodilating pain syndrome (table 5).—Subcutaneous administration of histamine was considered a failure in 3 cases. One of these patients obtained relief for one month. One patient was subsequently relieved by nicotinic acid, and 1 was not.

Nicotinic acid given subcutaneously produced good results in 3 cases. Doses of 200 mg. of nicotinic acid daily were required in 1 case before relief was secured. In 1 of the 3 cases in which subcutaneous administration of nicotinic acid was considered a failure, histamine given subcutaneously also failed to give relief. The patient was completely symptom free while receiving 5 cc. of the extract of the whole adrenal cortex as prepared by E. C. Kendall plus 1 cc. of 1:50,000 solution of epinephrine.

Vasodilating pain syndrome associated with perennial vasomotor rhinitis (table 5).—Histamine given subcutaneously failed to give relief in 3 cases. In

1 of these cases intravenous treatment with histamine also failed. In 2 of the cases in which treatment with histamine was classed as a failure relief for two weeks was followed by recurrence. Nicotinic acid gave complete relief for one month in the 1 case in which this type of treatment was considered a failure.

Vasodilating pain syndrome associated with exacerbations of myalgia of the head (table 5.)—Of the 2 patients for whom subcutaneous administration of histamine was considered a failure, 1 was relieved of symptoms for six weeks by this means and the other obtained no relief and also failed to obtain relief from niacin. In the 1 case in which treatment with niacin was considered a failure treatment had to be stopped because of nausea and vomiting following injection.

Vasodilating pain associated with exacerbation of myalgia of the head and perennial vasomotor rhinitis (table 5).—In 2 of the 3 cases in which treatment with histamine was considered a failure symptomatic relief for two weeks and in 1 case for one week was obtained.

Myalgia of the head associated with perennial vasomotor rhinitis (table 5).—In 1 of the 3 cases in which histamine administered intravenously failed to give relief, 0.1 cc. of 1:100,000 solution of histamine base produced a marked increase in symptoms ten minutes after injection on three occasions. No flushing of the skin could be observed.

In 1 of the 16 cases in which nicotinic acid given subcutaneously produced good results histamine given intravenously had produced marked increase in headache one hour after injection. In both cases in which treatment with nicotinic acid was a failure subcutaneous injection of histamine also failed to give relief.

Myalgia of the head (table 5.)—Histamine given subcutaneously failed to give lasting relief in 4 cases, in 1 of which, however, symptoms were relieved for one month. In 1 of the 13 cases in which subcutaneous injection of nicotinic acid gave good results abdominal cramps were produced by 100 mg. doses but relief was maintained by 50 mg. daily; in 2 of the 6 cases in which this type of treatment was considered a failure relief of symptoms was obtained for two weeks.

Comment.—It was concluded from the results obtained that histamine and nicotinic acid were equally effective in the treatment of vasomotor rhinitis. The good results reported by Harris and Moore in the treatment of Ménière's syndrome with nicotinic acid were confirmed. From the small number of cases in the series in which Ménière's syndrome was the only complaint, no comparison could be made between the effectiveness of histamine and nicotinic acid in relieving symptoms.

The only conclusion that may be drawn is that both histamine and nicotinic acid administered as described herein are relatively useful drugs in treating the physical allergy syndrome. From the results obtained one may conclude that the effect is obtained by fundamentally the same biologic mechanism. The only contrary evidence obtained was in the 2 cases in which good results were obtained by the use of nicotinic acid and histamine in conjunction after each had failed to produce a good result when used separately. I am unable to explain this observation.

Treatment with nicotinic acid amide.—An attempt was made to see if treatment with nicotinic acid amide would give results similar to those obtained with niacin (nicotinic acid). Sixteen patients were treated.

To this group 100 mg. of nicotinic acid amide were given subcutaneously daily for five days. The group consisted

of 5 cases of vasomotor rhinitis, 10 of myalgia, and 1 of vasodilating pain. No improvement in any of these cases was obtained with this treatment.

In a successful result with the treatment of vasomotor rhinitis with nicotinic acid, objective changes in the nasal mucosa are often observable within twenty-four hours after the optimal dose is reached. The results are somewhat slower in cases of myalgia and vasodilating pain of the head but in a successful case improvement in symptoms always occurs within seventy-two hours of reaching the optimal dose. The lack of response in the 11 cases of vasomotor rhinitis to nicotinic acid amide (niacin amide) suggests that the improvement is associated with the vasodilating effect of nicotinic acid which is absent in the nicotinic acid amide.

The number of patients treated is too small for the results to be conclusive but it seems improbable that nicotinic acid amide given in doses comparable to those which had proved effective for nicotinic acid, could have failed to produce a single favorable response if nicotinic acid amide were of benefit in this type of case or if the benefit obtained by administration of nicotinic acid were purely psychogenic.

Treatment with the extract of the adrenal cortex. — Because a review of the literature suggested that the abnormal cells involved in the physical allergy syndrome behaved as though they were deprived of cortical hormone, Dr. E. C. Kendall kindly supplied me with his extract of the whole cortical substance for clinical trial. A preliminary report of the results obtained is appended.

Seven patients who had vasomotor rhinitis were given 5 cc. of the extract of whole adrenal cortex twice daily for three days without provoking any observable response. This is in accord with the observations of Prickman and Koelsche[67] in the treatment of asthma. Nine patients who had vasomotor rhinitis were given twice daily injections of 0.1 cc. of a 1:10,000 dilution of epinephrine for three days. Eight presented no objective or subjective change in the nasal signs or symptoms. One patient who had a peculiar combination of vasomotor rhinitis and migraine secured both objective and subjective relief from epinephrine and was subsequently maintained symptom free with nicotinic acid.

Fifteen patients with vasomotor rhinitis were given 5 cc. of Kendall's extract of whole adrenal cortex plus 0.1 cc. of a 1:10,000 dilution of epinephrine twice daily. Marked diminution in the swelling of the nasal mucosa together with definite return toward the normal pink color of the membranes was observed in 14 of these cases. There was also a marked reduction in the symptoms of nasal congestion, sneezing and watery discharge.

These results suggest both that there is a synergistic action between the hormones of the adrenal cortex and medulla and that an insufficient supply of these hormones may be present in the cells reacting in the physical allergy syndrome.

Control series. — The 16 cases in which nicotinic acid amide was used and 15 cases in which injections of adrenal cortical extract or epinephrine were used without effect give a control series of 31 cases which may be compared to the group receiving either histamine or nicotinic acid.

COMMENT

If the four symptom complexes presented in this paper are assumed to be representatives of an integrated group of symptoms, having as a basis physical allergy of the head, the facts gleaned from the study of one of the members of the larger symptom complex then may be correlated with the other members. If

this assumption is correct, then the control of water and electrolyte balance should be of some value in the treatment of vasomotor rhinitis, myalgia and the vasodilating pain syndrome as it is in Ménière's syndrome. The importance of the control of permeability and the transfer of inorganic ions and water has been demonstrated in the case of Ménière's syndrome by Mygind and Dedering,[64] Furstenberg, Lashmet and Lathrop,[28] and Talbott and Brown and others.[84,85] It does not seem unreasonable to assume that the disturbances of ionic balance and permeability which have been demonstrated for one member of the physical or intrinsic allergy complex are present in other members of this group of conditions since symptoms and signs which seem to depend on disturbances of metabolism and permeability of cell membranes are found in the entire group. That this is true is suggested by the empiric use of potassium salts in these conditions for years.

It can be assumed that sensitization to physical agents may take place and may occur more readily in the presence of infection. The clinical history, especially in cases in which myalgia of the head and vasodilating pain syndrome have followed extreme exposure to cold or have developed during an acute or chronic infection, reinforces the evidence for association of infection with sensitization. The good results obtained occasionally in these disorders by elimination of foci of infection may be on the basis of the elimination of a sensitizing factor.

These factors suggest that better clinical results might be obtained by combining several methods of therapy, such as the giving of potassium salts, and dehydrating agents in combination with histamine or nicotinic acid. This should be the subject of further clinical investigation.

SUMMARY AND CONCLUSIONS

In this paper I have shown that many of the head pains associated with nasal and aural disease are on the basis of a physical allergy rather than being due to the infection syndrome.

In considering vasomotor rhinitis, Ménière's syndrome, myalgia of the head and the vasodilating pain syndrome as interrelated components of a syndrome of physical allergy it is necessary to show that they are interrelated, that they are allergic, and that the type of allergy involved is the intrinsic or physical type. This I feel has been done.

The allergic nature of perennial vasomotor rhinitis has been demonstrated so often that it now is generally accepted. The lack of relief secured by the usual allergic treatment seems to lie in the failure to distinguish between immunologic and intrinsic allergy. Most recent students of Ménière's syndrome feel that in all probability the disease has an allergic background. They may have been confused by the fact that clinically significant skin tests have not been obtained in cases of Ménière's syndrome and because other allergic manifestations were not found. This confusion seems to have arisen because of the existence of two symptom complexes with many similar symptoms, but with differing biologic mechanisms; ordinary allergy of the immunologic (antigen antibody) and intrinsic or physical allergy in which an antigen antibody type of reaction does not seem to be present. Many of the difficulties in considering Ménière's syndrome as an allergy are cleared up by considering it as frequently an example of "intrinsic" allergy. The statement that Ménière's syndrome is not associated with other manifestations of physical allergy I feel to be incorrect, and I believe that further careful observation will substantiate this.

Myalgia and the vasodilating pain syndrome have not won general acceptance

as "manifestations" of allergy and therefore I have presented at some length the evidence from the literature tending to substantiate this viewpoint as well as that in regard to the confusing pain references in these conditions. I have also reviewed the work of Lewis on the sensitization of tissues to physical agents, a possible mechanism through which sympathetic stimuli, both objective and subjective, can set off a train of allergic symptoms. The response of these conditions to treatment both with histamine and nicotinic acid as well as their definite association with perennial vasomotor rhinitis are additional reasons for considering myalgia and vasodilating pain syndrome as allergic phenomena.

The interrelation of the symptoms of the intrinsic allergy complex is well brought out by the occurrence of vasomotor rhinitis in the vasodilating pain syndrome. Its presence is considered one of the diagnostic features of the condition. The simultaneous occurrence of vasomotor rhinitis and myalgia is also common. Halle and his associates have emphasized the association of Ménière's syndrome and myalgia of the head. I have observed 4 patients who have had all four components of the "intrinsic allergy" syndrome; these were precipitated synchronously by the same physical stimulus, relieved by the same treatment and precipitable by appropriate doses of histamine and nicotinic acid. I believe that further clinical observation will confirm the interrelation of the four components of the "intrinsic allergy" syndrome, namely, vasomotor rhinitis, Ménière's syndrome, myalgia of the head and the vasodilating pain syndrome. The evidence has also been given which supports the idea that so-called histaminic cephalgia is not unique but is merely an outstanding example of the vasodilating pain syndrome.

I cannot refrain from quoting as a conclusion to this paper the remarks of that pioneer Minnesota physician, Dr. J. E. Finch, made at the "Symposium on Myalgia" before the Minnesota State Medical Association in 1877: "To collect facts and endeavor to discover the principles which underlie and determine them is the noblest of mental effort. This is called theorizing and is particularly distasteful to some persons who seem to forget that without theory there is no science. In the theory here submitted, which is quite different from anything we have seen on the subject, we have endeavored to make use of facts or well recognized principles with the introduction of as little pure hypothesis as possible. We are not so vain as to pretend that it is complete in every aspect, but even if our reasoning be unsound and our conclusion fallacious, some of our brethren, while pointing out our fallacy, may alight upon the truth and thus our effort will not have been in vain."

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DISCUSSION

FRENCH K. HANSEL, M.D., St. Louis, Mo.: Dr. Williams has thrown a great deal of light on the four components of the so-called syndrome of intrinsic allergy. In allergy circles there is a definite distinction made between what is known as extrinsic allergy, that is, which is caused by pollen, dust and foods, and intrinsic allergy. Many of the cases of so-called vasomotor rhinitis and asthma, in which no definite cause can be determined are placed in the intrinsic group. On the other hand, the fundamental pathology is the same. There is marked eosinophilia of the tissues, sometimes even more pronounced than in the extrinsic cases.

It seems to me that the fundamental mechanism involved in all of these syndromes is very much the same. One might say that the mechanism involved in Ménière's syndrome, whether it is associated with definite allergy or not, has the same fundamental factors concerned in its cause. In the case, for ex-

ample, of allergy of the parotid gland a very interesting phenomenon occurs. There is no edema of the gland, but there is formation of a thick, mucous plug which obstructs the duct. Under microscopic examination is shown a dense infiltration of the mucous plug with eosinophils.

Secretion from the parotid gland is produced by stimulation of the parasympathetic fibers of the cranio-sacral autonomic system. Stimulation of the parasympathetic fibers produces the thick mucous plug mentioned above. So it seems to me in all these cases there is an imbalance in the sympathetic and parasympathetic factors. We have had many cases of vasomotor rhinitis and some cases of asthma due to dust or to foods, in which some of these other syndromes occurred, such as headache. We found that by treatment with histamine the patients got relief from headache. In some cases the nasal symptoms were relieved by treatment by allergy methods, but the headache was relieved only by the use of histamine. We have done the same thing with nicotinic acid. In Dr. Williams' cases, it seems to me, nicotinic acid and histamine produce stabilization of the autonomic mechanism which is imbalanced in allergy. Good results follow in those cases in which autonomic balance can be restored. It can be restored in specific allergy by treatment by allergy methods. I feel that the use of these drugs has definite value as a supplementary treatment in some cases and as a means of relief in those cases in which other methods have failed.

NOAH FOX, M.D., Chicago, Ill.: The confusion existing in the classification and description of headache has been due to our meager knowledge of the mechanism of these pains, as well as the anatomy of the nerve distribution of the fibers involved. Often, in addition, there has been no close anatomic connection between the headache and the inflammatory condition of the nose, sinuses, or ears that are presumed to have caused it. As a result, the internist, the neurologist and the rhinologist has each protracted his version into the literature based on purely clinical observations or pathologic studies. Though in many of these conditions the allergic nature is apparent because of other allergies in the individual or his family, great difficulty exists in proving the allergic status in those patients suffering from an intrinsic type of allergy, such as the bacterial or endocrine type.

Dr. Williams has formulated an hypothesis which considers vasomotor rhinitis, Ménière's symptom complex, myalgia of the head, and vasodilating pain syndrome interrelated conditions representing a manifestation of intrinsic or physical allergy. He has accumulated an unusual and excellent amount of basic physiologic and pharmacologic evidence developed by investigation of conditions which Dr. Williams has felt are comparable to the syndrome in question. It would appear from this evidence that the mechanism of the above syndromes might be explained on the theory of an intrinsic or physical type of allergy. Pathologically these conditions represent hyperplasia, a condition always present where allergy has existed for any length of time. However, chronic suppuration is also capable of causing hyperplasia. According to Dr. Williams' thory of an intrinsic etiology, he must concede both an allergic and bacterial factor present.

To this assumption I should like to add further evidence. In 1937 I reported the results of the following experiment: Small pieces of tissue were collected aseptically at operation from the antrums of 25 asthmatic patients, whose antrum washings had constantly proven sterile. One portion of the tissue was stained by Giemsa's method, while another portion was implanted into the peritoneal cavity of guinea pigs. After forty-eight hours the implanted tissue was removed, fixed, sectioned and stained by Giemsa's method. Of the 25 specimens of tissue implanted all without exception revealed a heavy, mixed bacterial growth, coccal forms predominating. Of the 25 specimens of tissue prepared but not implanted, only 8 cases demonstrated organisms. The organisms identified were streptococci, staphylococci, Friedlander's bacilli and diphtheroids. No organisms of the gastro-intestinal flora were encountered, which might tend to prove absence of contamination from the bowels. This experiment demonstrates the presence of bacteria constantly in allergic tissue, even though washings are negative. The effect of these organisms or their toxins deep in the tissues might be the factor responsible for the conditions in question, and if so, would be considered an intrinsic mechanism.

There can be little doubt that in numerous cases of Ménière's disease, an allergic picture is demonstrable, both by history and skin testing. On the other hand certainly allergy, neither intrinsic nor extrinsic, appears as a factor in those patients where angiopathic disease is clearly present, as in arteriosclerotics, etc.

For years, because of the absence of skin reactions elicited in vasomotor rhinitis, we

assumed an intrinsic factor to be causing the condition. I might add that the terms "intrinsic and extrinsic," as Dr. Hansel has mentioned, are terms used by general allergists merely as pigeonholes for those conditions in which they are unable to get positive skin tests. As a matter of fact, it might also be said that it is an excuse for our lack of more precise methods of finding the etiology of some of these allergic conditions. We have found, for instance, that where we used more concentrated testing materials, many of the supposed bacterial types — and bacterial types are considered by the allergists as intrinsic allergies — are not intrinsic affairs at all. We lose track of the fact that allergy is actually a normal reflex slightly exaggerated. One individual has a different threshold of stimulus than another. One individual inherits a different nervous pattern than another. We all know that there are families that have headaches. There are allergic families, in which 2 or 3 of the individuals suffer from so-called vasomotor rhinitis, where 1 individual's predominant symptom is sneezing and where another's symptom is vasomotor obstruction. Those conditions are the same. One individual has gone on to inherit sneezing nerves and the other one has gone on to inherit nerves which control the arterial system of the nose.

When Dr. Williams said that we should not put hyperplastic rhinitis along with vasomotor rhinitis, I cannot agree with him. We have sectioned some 7,000 tissues, taken from people who have had various nasal allergic conditions, individuals who have had what we call vasomotor rhinitis, or hyperesthetic rhinitis. Some years ago we submitted many of these tissues to some of our best pathologists and the diagnosis given was "chronic inflammation" in all of them. The matter of degree, or amount of hyperplasia is the only difference in these tissues. It is merely a matter of the amount of change, and the relationship between the water and solid tissue balance.

Dr. Williams theorizes that certain substances are produced in the muscles of patients with myalgia, which substance might be histamine, particularly since the muscle pain is of a pulsating character. This, however, is difficult to prove, since injection of histamine into the muscles, even in doses large enough to cause headache and severe fall of blood pressure does not give rise to any sustained muscle pain or myalgic node.

Dr. Williams is to be congratulated for his unusual treatment of this most perplexing subject.

MILES ATKINSON, M.D., New York, N. Y.: The test to which Dr. Wililams has tagged my name is not the test that I have described. He used 0.1 cc. of a 1:1,000 solution of histamine base. Is that correct? I used 0.1 cc. of a 1:20,000 solution of histamine base.

HENRY L. WILLIAMS, M.D., Rochester, Minn.: Dr. Hansel's statement that he has been able to help some cases of headache associated with specific allergy by treatment with histamine or nicotinic acid is interesting, because in the group of specific allergies I have had very few successes with the use of either histamine or nicotinic acid.

In regard to Dr. Fox's discussion, I excluded asthma from the "syndrome of intrinsic allergy." As a matter of fact, with the doses of histamine and nicotinic acid that I used, asthma invariably was not benefited or was made worse.

Dr. Fox seemed to misunderstand me in regard to the association of vasomotor rhinitis and hyperplastic sinusitis. Instead of stating that they should be divided I said they should not be divided as they are fundamentally the same condition with a superadded infection in the case of hyperplastic sinusitis. The pathology of vasomotor rhinitis and hyperplastic sinusitis, it seems to me, is essentially edema rather than hyperplasia. The hyperplasia is secondary to long standing edema. I believe this indicates that there is some disturbance of cellular permeability which is controlled by adrenal hormones.

I am unable to accept Dr. Fox's statement that bacterial allergy is intrinsic or physical allergy, or the statement that these types of allergy are distinguishable by the presence or absence of positive skin tests. In specific allergy an antigen antibody type of mechanism is present; in intrinsic or physical allergy such a mechanism has not been shown to be present. Since an antigen antibody type of reaction is probably present in bacterial allergy, this should be included in the group of specific allergies.

TABLE I.

Comparison of Symptoms of Various Components of Sluder's Syndrome with the Symptoms of the Various Components of the Syndrome of Intrinsic Allergy

Conditions	Sluder's Syndrome	Vascular Headache; Histaminic Cephalgia	Myalgia of Head	Vasomotor Rhinitis	Hyperplastic Sinusitis*	Meniere's Syndrome
Background of sensitivity to cold	Present	Occasional	Present	Present	Present	Present
			PAIN			
Underlying muscular tenderness		Occasional	Characteristic	Occasional	Frequent	Often present
Unilateral or bilateral	Unilateral but may shift sides	Usually unilateral	Unilateral usually; may be bilateral	Occasional; unilateral or bilateral	Unilateral but may shift sides	Usually unilateral
Onset and termination	Sudden or slow	Sudden	Both slow	Slow	Usually slow, may be sudden	Same as myalgia
Character	Somatico-visceral	Somatico-visceral	Somatico-visceral	Somatico-visceral	Somatico-visceral	Same as myalgia
Controlled by arterial pressure	Not stated	Yes	Yes, in "attacks"	Yes, when present	Yes, when present	Same as myalgia
Occurrence at night	Not mentioned	Usually worse at night than day	About 50%	Occasionally	Often	Same as myalgia
Reproduced by histamine	Never tried	Yes	Yes	Yes	The vasomotor component	Same as myalgia
Arterial tenderness	Not tried	Yes	Yes	Occasional	Occasional	Infrequent
Palpation reference	Not noted	None	In height of attack	When myalgia is present	When myalgia is present	Infrequent
Effect of vasodilators (cholinergic)	Not noted	Relieves	Relieves attack	Relieves paroxysm	Relieves vasomotor rhinitis component	Infrequent

*Hyperplastic sinusitis is perennial vasomotor rhinitis with secondary sinusitis.

TABLE I.—(Continued)

Comparison of Symptoms of Various Components of Sluder's Syndrome with the Symptoms of the Various Components of the Syndrome of Intrinsic Allergy

Conditions	Sluder's Syndrome	Vascular Headache; Histaminic Cephalgia	Myalgia of Head	Vasomotor Rhinitis	Hyperplastic Sinusitis*	Meniere's Syndrome
SYMPTOMS REFERABLE TO EAR						
Tubotympanitis	Frequent	Not reported	Occasional	Frequent	Frequent	Occasional
Mild vertigo on change of position	Frequent	Not reported	Frequent	Occasional	Common	Frequent
Severe vertigo (Meniere's)	Occasional	Occasional (origin of treatment for Meniere's)	Occasional	Occasional	Occasional	Characteristic
Tinnitus	Frequent	Occasional	Frequent	Uncommon	Occasional	Characteristic
Deafness	Not reported	Occasional (labyrinthine type)	Occasional (labyrinthine type)	Occasional (tubal occlusion)	Occasional (tubal occlusion)	Characteristic
SYMPTOMS REFERABLE TO THE EYE						
Conjunctival injection	Present	Present, usually unilateral	Frequent, unilateral or bilateral	Usually bilateral	Usually bilateral	Occasional
Lacrimation	Frequent	Present and usually unilateral	Occasional, unilateral or bilateral	Frequent, usually bilateral	Frequent, usually bilateral	Occasional
Edema of eyelids	Occasional	Common	Occasional	Frequent	Occasional	Occasional
Dilatation or narrowing of pupils	Occasional	Occasional	Occasional	Occasional	Occasional	Occasional

TABLE I.—(Continued)

Comparison of Symptoms of Various Components of Sluder's Syndrome with the Symptoms of the Various Components of the Syndrome of Intrinsic Allergy

Conditions	Sluder's Syndrome	Vascular Headache; Histaminic Cephalgia	Myalgia of Head	Vasomotor Rhinitis	Hyperplastic Sinusitis*	Meniere's Syndrome
SYMPTOMS REFERABLE TO THE NOSE						
Congestion	Present	Usually unilateral	Frequent; unilateral or bilateral	Invariable	Invariable	Occasional
Polypoid hypertrophy	Present	Absent	Absent	Frequent	Invariable; usually bilateral	None
Thin discharge	Present	Present, usually unilateral	Occasional; unilateral or bilateral	Invariable	Common	Occasional; usually unilateral and on side of affected ear
Itching and burning of skin	Present	Occasional	Occasional	Frequent	Occasional	Not noted
SYMPTOMS REFERABLE TO MOUTH AND THROAT						
Glossodynia	Present	Not described	Occasional	Occasional	Occasional	None
Pain referred to ear	Present	Present	Present; superior constrictors involved	Rare	Rare	None
Itching of palate, tonsillar pillar	Present	Not noted	Rare	Common	Common	Not noted
Globus	Present	Not noted	Not unusual	Occasional	Occasional	Not described
Hoarseness	Present	Not noted	Arytenoideus involved	Occasional	Rare	None
Pain on swallowing	Present	Not noted	Superior constrictors involved	Rare	Rare	None

TABLE II.

Diagnoses Made in 362 Cases of Intrinsic Allergy Syndrome

Diagnosis	Cases
Perennial vasomotor rhinitis only	173
Vasomotor rhinitis* and myalgia	30
Vasomotor rhinitis*, myalgia and vasodilating pain	13
Vasomotor rhinitis* and vasodilating pain	13
Vasomotor rhinitis* and Meniere's syndrome	11
Vasomotor rhinitis*, Meniere's syndrome and myalgia	8
Vasomotor rhinitis*, myalgia, vasodilating pain and Meniere's syndrome	2
Myalgia, Meniere's syndrome and vasodilating pain	23
Myalgia	36
Myalgia and Meniere's syndrome	16
Myalgia and vasodilating pain	5
Vasodilating pain	10
Meniere's syndrome	22
TOTAL	362

*In 75 cases vasomotor rhinitis was associated with other disorders.

TABLE III.

Results of Atkinson Test and Treatment with Histamine and Nicotinic Acid Amide

Diagnosis	Results of Test	RESULTS OF TREATMENT		
		Nicotinic Acid Used in 49 Cases		
		Good*	Fair*	Failure
Meniere's Syndrome	Positive	8		1
	Negative	4		
Myalgia and Meniere's syndrome	Positive	1		
	Negative	3		
Vasomotor rhinitis and Meniere's syndrome	Negative	1		
Vasomotor rhinitis	Positive	9	3	2
	Negative	6		1
Myalgia of head	Positive	2		1
	Negative	1		3
Myalgia and vasomotor rhinitis	Positive	2		
	Negative	1		
Diagnosis	**Results of Test**	**Histamine Used in 6 Cases**		
Vasomotor rhinitis	Positive	Fair		
	Negative	Very good		
	Negative	Good		
	Strongly positive	Very good		
Myalgia	Strongly positive	Failed		
Vasodilating pain	Negative	Failed		

*A good result was considered one in which complete or nearly complete relief of symptoms was maintained for 3 months or more. A fair result was considered one in which 50 to 90 per cent relief was maintained for 3 months or more.

TABLE IV.

Results of Intracutaneous Test and Treatment with Histamine in 55 Cases

Diagnosis	Total Cases	Histamine Skin Test	RESULTS OF TREATMENT		
			Good	Fair	Failure
Vasomotor rhinitis	28	Positive	5	1	10*
		Negative	6		6†
Meniere's syndrome	1‡	Positive			1
Myalgia	14	Positive	5	1	3
		Negative	4		1
Myalgia and vasomotor rhinitis	4	Positive	3		1
Vasomotor rhinitis and vasodilating pain	7	Positive	3	1	2
		Negative	1	1	
Vasodilating pain	1	Positive		1	

*Subsequent relief with niacin in 1 case and with simultaneous use of histamine and nicotinic acid in 1 case.

†Two patients had asthma. Subsequent relief with niacin in 2 cases.

‡Also positive Atkinson Test.

TABLE V.

Results of Treatment with Histamine or Niacin in 327 Cases

Condition	HISTAMINE			NIACIN		
	Good	Fair	Failures	Good	Fair	Failures
Perennial vasomotor rhinitis	33 (55%)	7 (14%)	18 (31%)	54 (57%)	10 (11%)	30 (31%)
Perennial vasomotor rhinitis and Meniere's syndrome	0		2	8		1
Perennial vasomotor rhinitis, vasodilating pain, Meniere's syndrome and myalgia	1*			3		
Vasomotor rhinitis, Meniere's syndrome and myalgia	0		2	8		1
Meniere's syndrome				16	3	1
Meniere's syndrome and myalgia	2		2	10	3	2
Vasodilating pain syndrome†	2		3	3		3
Vasodilating pain syndrome and perennial vasomotor rhinitis	4		3	6	2	1
Vasodilating pain and myalgia	1		2	2		1
Vasodilating pain, myalgia and perennial vasomotor rhinitis	6		3	2	1	2
Myalgia and perennial vasomotor rhinitis	9		3	16		2
Myalgia	9	1	4	13		6

*Treatment was changed to niacin by mouth after 6 months with continued relief of symptoms.

†Histamine was given intravenously in this group of cases.

THE PRESENT STATUS OF EYE EXERCISES FOR IMPROVING VISUAL FUNCTIONS

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ONE OF the fundamental laws of biology is that repetition of an act facilitates its performance. The first time one does something it may be done clumsily and laboriously, the hundredth time it may be done dexterously and with ease. No principle of physiology and psychology is more firmly established than this.

It is, of course, the basis of acquiring skill in any performance. To understand its modus operandi one must recall the way the nervous system is organized and how it operates. There are a prodigious number of nerve cells and an intricate network of communicating fibers. These fibers afford pathways for messages — nerve impulses — from one "center" or group of nerve cells to another, and finally to a muscle or gland or other effector, unless somewhere along its course it is arrested by inhibitory forces. When an act is clumsily done the messages are not passing smoothly and freely along the proper pathways; false turnings have to be corrected before the desired end is reached. If the act is repeated over and over again the passage of the nerve currents in the proper pathways is made easier and more efficient, i.e., with fewer false turns or hesitations, "facilitated" as it is called. This statement simplifies the matter of learning too much. It would result in rigidity, lack of flexibility. If repetition, by facilitating the passage of messages along a fixed route, were the whole story, how could one account for the very important fact that when one has learned how to do something he is able to adapt the performance to unexpected variations in conditions which may arise and may call for immediate changes in the way the act is best performed in some of its details?

Repetition is the essential feature of exercises, training and learning as these terms are used here. Obviously, repetition of an act with a faulty method will serve to confirm the fault just as surely as repetition with a correct method will confirm the good habit. This is where the capable coach or trainer or teacher watches early for faulty performance.

For instance the golfer who practices his swing in a faulty way only confirms his faults. He will improve and, if he has some natural talent, may consistently get below 100, but unless he has a competent teacher to watch and correct him, he can not expect to break 80 except by a rare streak of luck. His play will be erratic and uncertain. So in orthoptics, in doing bar-reading, the more the patient reads in the wrong way, shifting his fixation from eye to eye so swiftly perhaps that he reads aloud smoothly line after line, the more deeply rooted does his suppression become. Many other examples could be cited by the experienced orthoptist of ways in which exercises, perhaps with automatic electric motor contrivances, cause the patient to make certain movements but do not guard against his doing them by a faulty method.

If the act involves muscular action, as most acts do, one result is that the muscles involved increase in size and

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power (hypertrophy), become stronger. For example, in learing to row two things are involved, skill in handling the oars, and strong enough muscles to do the work required. If the muscles are already strong enough no increase results from the exercise.

There are many activities which call for increased strength of the muscles as well as increased skill in using them. This is notably the case if the learning of a given skill is undertaken before the individual has reached full muscular development. If a boy of 12 takes up golf he can learn to swing the clubs with great skill and graceful coordination but until his muscles become stronger he cannot strike the ball hard enough to make a long drive. So the man who is strong will say the secret of a long drive is the skill and coordination, while the weak-muscled player will say it is the power you can put into it that makes the ball go a long way. Of course, both skill and power are essential. In the case of ocular movements there are still many who think it is a matter of strength, of muscular power that is lacking in, for example, convergence insufficiency. Others, realizing the very great excess of muscular strength that is provided in the ocular muscles, stress the lack of coordination, the inability to use the muscles, the lack of "skill."

That exercise and practice improve the performance of muscular or motor activities no one is likely to deny. When it comes to sensory activities there may be doubts in the minds of some. How, they might ask, will practice improve sight of hearing? The dioptric images formed on the retina are the stimuli. The only way to improve sight is to get a better image on the retina. This cannot be obtained by practice or exercises. This conception of seeing is very far from correct. The first step in seeing is having a good image on the retina and, by fixating, on the best part of the retina, the fovea; but this is *only the first step*.

It is thoroughly well-established that it is impossible to experience a pure, isolated sensation. There are two groups of factors which affect all sensations as they reach consciousness and become perceptions. In the first group are all those other sensations coming into consciousness at the same time not only through the visual apparatus but also through the tactile, kinaesthetic, hearing, and all other avenues. The second group comprises memories of past experiences and even inherited predispositions. This is of enormous practical importance in understanding how one sees. It explains how a mere glimpse of an object well known from previous experience results in "seeing" many details which as a matter of fact are supplied by memory. For example, the apparent color of a dress may be altered by the color of the lighting under which it is seen, but because one knows from many past experiences what the color is under daylight (white light), the object is "seen" as having its well known, not its apparent color.[7] So in even greater degree with apparent size, distance and form. The pattern as a whole is appreciated as a unitary and complete pattern without any conscious analysis of its individual details. Different stimuli, often apparently contradictory, may produce the same response. A square card seen obliquely makes an image on the retina which is not square, but it is "seen" as square, from whatever angle it is viewed. "The essential factor, therefore, in the emergence of any pattern is its *meaning*."[8]

The memories of past experiences (usually unconscious) affect profoundly the various sensations. Exercises and practice serve to build a substratum of experience, which facilitates perception of whatever is the subject of the practice and exercises. The effects of exercises which I wish to stress are: first, the

facilitation of pathways and of conditioned reflexes; second, the accumulation of a fund of experiences and associations from which are evoked, by the immediate sensations, responses in the brain which lead to interpretations and which altogether build up and determine the perception by consciousness. Seeing, for example, is only half ocular, the other half is cerebral. One cannot say that either one is the more important since both are equally indispensable and in practice inseparable. The third, which I wish not to stress but to minimize, is the increase in muscular strength.

Some examples of the effect on sight of practice or exercises:

I. Treatment for color blindness of men rejected from military service. The usual procedure as described by optometrists consists in the fixation of colored sources of light, for several minutes, and the use of filters, with daily check of progress on the pseudo-isochromatic plates. The "Light and Filter" treatment.

Two instructors in optometry at Ohio State University, Charles S. Bridgman, Opt.D., Ph.D., and Henry W. Hofstetter, Opt.D., Ph.D., skeptical about this method of improving color vision, tested several subjects 3 of whom had passed the Navy tests successfully after treatments by a practicing optometrist.[3] They concluded that their results did not in any way support the assumption that the exercises intended to train color vision are of any assistance. It appeared on the contrary that the exercises are merely time-consuming, that the improvement in scoring is related only to the *learning made possible by repeated testing*. The following is a sample of the results obtained. A color blind subject from the eye clinic tested in the usual way scored 15 correct out of 46 plates. He was tested four times. He was told when he was wrong but was not told the correct answer. His scores were 27, 35, 43 on the second, third and fourth tests. Similar results were shown by other subjects.

The authors further concluded that "those who criticize the practice of training color vision are entirely justified." They "find no evidence that color filter treatment is effective in improving color blindness." They "question the type of evidence which has been accepted as conclusive indication of color vision improvement"; "that this proof can be supplied only by measurement of fundamental color vision functions" (Rayleigh equation, etc.). The treatment, they said, is not justifiable since it may lead to the acceptance of responsibilities which the patient cannot fulfill though he erroneously believes himself cured of color blindness.

II. Studies of Peripheral Vision by Frank N. Low.[13] Over 200 subjects were tested under properly controlled conditions. What we are interested in is that "the evidence indicates that peripheral visual acuity can be trained." Twenty per cent of the original group of 100 when retested showed an improvement of 16 per cent. The improvement was roughly proportional to the amount of time taken by the test.

III. Studies of speed in seeing a number of digits or letters. Renshaw[16] reported work on the Polish lightning calculator, Dr. Finkelstein, who memorized:

8 digits in .003 seconds
12 digits in .824 seconds
16 digits in 1.73 seconds

Renshaw then trained a dozen university students, 2 of whom were able after practice to surpass Finkelstein's record for speed in learning digit groups. He remarks: "The difference between the expert and the novice in the rapid and accurate perception of visual material is the same difference between the expert and the novice in the performance of any skillful act. We have to learn

to see just as we have to learn to swim, to play the piano, or to speak French. This can be done with skill and efficiency or it can be done haltingly and ineffectively. When we train children to learn to spell English words by replacing the wasteful and ineffective disjunctive method of seeing words with the proper method of visual perception, spelling difficulties disappear. Not only does a child spell accurately and easily, but he comes to enjoy spelling. An incidental by-product also is that his rate of reading and index of comprehension automatically show a corresponding improvement."

IV. Another example of learning to see is afforded by having a person with practically normal eyes wear eikonic lenses magnifying one meridian of one eye. This throws his binocular space perception out of gear but does not affect his monocular judgments of space. For a few days, especially in the morning, he is more or less annoyed or upset by this, but as time goes on he learns to suppress his faulty binocular perceptions and rely on his monocular clues. If he is placed in an environment where monocular clues are few or absent he is much disturbed, but when in situations presenting abundant monocular clues, a room for instance, he is aware of little or no effect on his seeing. *He has learned* to adapt himself to the new conditions.

V. Many examples could be cited from orthoptics of patients (with heterophoria) learning to see more correctly. For instance, a bookkeeper found his work laborious, slower than it ought to be with too many errors, and causing undue fatigue and frequent headaches. He had a considerable heterophoria with suppression which made it unduly difficult to follow lines across the paper correctly and difficult to add up columns of figures. A few weeks' training by an orthoptist and faithful homework resulted in teaching him to use his eyes more skillfully. His work was made easy and rapid, and fatigue greatly reduced.

VI. An example from heterotropia: A case of accommodative convergent strabismus with hypermetropia of 3 D and a small astigmatism, without glasses a convergence of 25 degrees, with glasses a convergence of 5 degrees (cosmetically straight). She was 12 years old. Her mother did not want her to wear glasses. A few months' training by an orthoptist resulted in the patient's learning to hold the eyes straight without glasses.

VII. A child with convergent strabismus and amblyopia of the left eye first learned to see with the amblyopic eye by occluding the right eye; next learned simultaneous perception, fusion, stereoscopic depth perception on the synoptophore and finally good binocular vision off the instrument as a habit.

VIII. There is an important group of cases with true myopia, all of whom earnestly desire to get rid of their glasses. These patients have been told by ophthalmologists or optometrists that they must wear glasses all the time. They go to a follower of Bates who tells them to discard their glasses, which are crutches. To their surprise they find that nothing serious happens! Their sight of course is still blurred for distant objects but they find that by practice they acquire the ability to make out objects better through the blur than when they only temporarily took off their glasses during the glasses wearing period. They are apt to exaggerate this gain in talking about it.

IX. A young man who has been wearing weak concave lenses came asking if there was any way he could pass the 20/20 test. His vision with his glasses was 20/15 but without glasses was 20/30. He was given + 1.00 sphere O.U. to use constantly for three days. He then read 20/15 without glasses, 20/20 with + 0.50 sphere, some letters of 20/20

with + 1.00. This man was not myopic to begin with. He learned to relax his accommodation. He was judged myopic by someone who found that when – 0.50 sphere was placed before his eye he said, "That is better, clearer." If a young man who sees 20/15 sees it better with – 0.50 sphere it shows that he has good accommodation, not that he has myopia. Other evidence is necessary before that diagnosis is justified. He should read one or two *more* lines with the minus glass than he reads without.

In every one of the examples I have cited, the progress of the patient has depended on learning to use his eyes better. He learns to see the isochromatic plates better only by greater skill or proficiency in interpreting his visual sensations. The sensations are no better, he is still color blind, but he has learned to make so much better use of his defective apparatus that he has fooled the examiners. Another learns to see better with his peripheral retina, not because the sensations are better or the retinal images better, but because he has learned by practice to perform better the cerebral processes which are such an important part of seeing. The third learns to take in a group of figures in a small fraction of the time it took him at first, not because his visual sensations have improved but because he has learned how to use them by practice, by exercises. The man who experimented with aniseikonic lenses became adapted to them by practice, by learning to suppress what was not to his advantage to "see" and to utilize instead what he found to be dependable and therefore advantageous. That the bookkeeper with the heterophoria derived his relief from strengthening certain "weak" muscles, I stoutly deny. His muscles were not weak when he began and they were not notably stronger when he had recovered from his disability. His bad habits had been changed by skillfully directed exercises and practice to good habits. Probably he had been suppressing and that certainly cannot be cured by making a muscle stronger. The strabismus case with accommodative convergence learned to use her muscles so as to hold the eyes straight by *relaxing* certain muscles, not by *strengthening* them. The seventh case is the most beautiful demonstration of all. The amblyopia was cured by exercise, by practice, by learning to see. The patient was guided skillfully through the intricacies of binocular vision in its various stages to a complete cure. The eighth group shows how much can be gained by learning to see, even when the visual acuity is low, by persistent practice. Here belong the cases of improved speed and accuracy of recognition by practice when there is no question of improved visual acuity—the sailor, the airman, the hunter, the fisherman, the prospector all acquire by practice the ability to see better though the visual acuity remains the same. The last case shows how wearing a concave lens can induce such a tonic contraction of the ciliary muscle as to counterfeit myopia and how wearing a convex lens can induce relaxation by a few days' practice at learning how to see.

Use is made of the principles of practice and exercise in other branches of medicine, notably orthopedics where posture is corrected and various disabilities are relieved. Lately the Kenny treatment for infantile paralysis has received much publicity. It has achieved some brilliant results. The explanations given by the originator have been shown by competent investigators[14,19] to be false but that is not a sufficient reason for abandoning the treatment, the good points of which should be adopted.

Precisely similar comments can be made concerning the Bates treatment lately given much publicity by Huxley's book, by other books, and by numerous articles in the lay press.[1,2,4,10,12,17] The

number and prominence of these articles are evidence that the public is interested. Several editorials and reviews have appeared in the professional journals.[5,6,9,15,18] Notice was taken of the problem by the Committee on Optics and Visual Physiology at its last meeting in June 1943. It is not surprising therefore that this subject was selected as suitable for discussion by the Academy.

If one studies the various publications with an open mind, searching for the things which explain why the public wants it, one will be forced to admit that buried in a mass of what to us seem foolish gestures and performances best defined as hocus-pocus there are sound and fruitful ideas. (Duke-Elder refers to them as "Huxley's antics of palming, shifting, flashing, and the rest are probably as good treatment as any other system of Yogi or Coué-ism.") It is these that account for the survival and spread of the cult. It certainly is not foolish to believe that people can be taught to use their vocal organs in speaking or in singing, that they can be taught to play musical instruments, to dance, to skate, to play games like billiards or golf and, as a result of teaching, can learn to perform better than they could without instruction and training. In the same way, the public argue, they should be instructed in the use of their eyes. They consult the eye specialist and are given glasses but are given very little information about the nature of their trouble or about measures which might help them to use their eyes more successfully. Yet they hear of someone who went to a follower of Bates and got plenty of instruction. We all know that there are many patients who do not want to wear glasses and who would go to almost any length to escape that fate. We should take account of this in our dealings with such patients. Most of them appreciate having things explained to them. This takes time and patience but the doctor need not do the teaching himself; he may employ someone who can do it for him, after assuring himself that the patient will be given sound instruction. The patient of course cannot discriminate between the pseudo-science of the partly educated followers of Bates and the sound truths of the well-trained ophthalmologist.

However, it is important to point out that the hypothetical well trained ophthalmologist is often very ignorant of certain phases of the art and science of which he is supposed to be a master. He is apt to make the mistake of arguing that because Bates' theory of accommodation is incorrect his whole program is unsound. The history of medicine is one long list of tentative theories many of which later proved to be erroneous, but the facts they tried to explain remain firmly established though the theories were swept away. Quinine cured malaria and continued to cure it though the theories invented to account for it were entirely erroneous.

Among the important things emphasized by Huxley are: the value of relaxation and the harm of tension; the pernicious influence of fear, e.g., fear of light, fear of blindness, fear of using the eyes; the dual nature of seeing—sensation plus interpretation equals perception; and the necessity of giving attention and treatment to the cerebral side of the function. The fact that in carrying out these lines of treatment they indulge in mumbo-jumbo should not prevent us from making use of the good points concealed therein. It is often pointed out that Huxley's visual acuity has not improved in any extraordinary way. He admits that. The point is that he has *learned how to use what he has to better advantage*. It is not the primary retinal sensation that is improved; it is the neglected but vitally important cerebral part of seeing that has been trained.

Here are some quotations from Huxley: "——the art of seeing does not stand or fall with any particular physiological hypothesis." "The art of seeing is like other fundamental or primary psycho-physical skills, such as talking, walking and using the hands." "People may have the most fantastic views about physiology; but this will make no difference so long as their theory and practice of psycho-physical functioning remain adequate to their purpose. If psycho-physical skills depended for their development on a correct knowledge of physiology, then nobody would ever have learnt any art whatsoever. It is probable, for example, that Bach never thought about the physiology of muscular activity; if he ever did, it is quite certain that he thought incorrectly. That, however, did not prevent him from using his muscles to play the organ with incomparable dexterity."

With a more sound theoretical basis, and therefore more intelligent execution, the good results will be still more impressive. Orthoptics in its early days managed to cure certain of the conditions for which it is used by a more or less clumsy routine based on the theory that some muscle needed to be strengthened. With a more skillful technic based on a wiser, more rational theory the progress is far more rapid and sure in the hands of those who know how to apply these better methods.

To summarize: There is abundant evidence for the general proposition that exercises, repetition, practice, learning, lead to better performance, to the acquisition of skill. There are many eye conditions which exemplify this law. Since seeing is only partly a matter of the image on the retina and the sensation it produces, but is in still larger part a matter of the cerebral processes of synthesis in which memories play a principal role, it follows that by repetition, by practice, by exercises, one builds up a substratum of memories very useful for the interpretation of sensations, and facilitates the syntheses which are the major part of seeing. Also motor functions are perfected by practice, by learning, and reflex pathways are facilitated. Ophthalmologists have neglected this field and have concentrated their attention on the primary source of the sensation, the image on the retina, leaving to incompletely trained workers the cultivation of this field. The achievements obtained by the clumsy practices which have been developed should stimulate ophthalmologists to investigate and discover the valuable possibilities in this field which, I am convinced, await intelligent development. Finally, in assessing the value of exercises it must not be forgotten that many of these cases have a neurotic or psychic element and that such cases are often favorably affected by hocus-pocus. Do not make the mistake of thinking that this is the whole story.

I have said nothing about actual structural changes being brought about by exercises other than hypertrophy and hyperplasia of muscles and the changes thereby induced in tendons and perhaps bones.

Frankly I am very skeptical. I think a clearing of corneal opacities often occurs but not by practice or exercises. I doubt any changes in the shape or size of the eyeball brought about by such means. Permit me to quote Jesus who said: "Which of you by taking thought can add one cubit to his stature?"

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DISCUSSION

LAWRENCE T. POST, M.D., St. Louis, Mo.: As I knew several months ago that I was to open this discussion I have been accumulating my material since that time so my discussion is really of the subject and only one phase of it and not of Dr. Lancaster's paper, but I do want to say that I think his is a most thoughtful and excellent presentation.

There are few things that have contributed as much to human happiness as spectacles, primarily as a means of improving sight and secondarily for relieving eye strain. They do, however, have obvious drawbacks, though the one that is the least important, that of their detracting from good looks, is probably the most widely inhibiting factor to the wearing of them. Consequently everyone would like the good effects of glasses without the glasses.

At this time the incentive for good vision without glasses is particularly keen in the group of young men and women who desire appointments in the Armed Forces, because the regulations for officers require a high standard of vision without correction by lenses.

For many years various efforts have been made to acquire the good effects of glasses by other means. The idea that exercise of the ocular muscles might accomplish this has long been advocated by some. Among these was a New York doctor who some twenty years ago elaborated a series of exercises for which he claimed great things in the improvement of vision. His ideas were based on loose experiments and speculations that were in the main contrary to those generally accepted by scientists, and his method of approach to the subject was distinctly unorthodox. Hence, scientists universally rejected his methods in toto. However, there were others who persisted in giving the type of training that he advocated and claimed good results.

A ready explanation for this exists in several facts, namely: there are many wearing glasses who do not need them; there are some whose visual disabilities are due to muscular irregularities and for whom suitable ocular exercises are truly valuable; and there are those who need psychological help in the use of the eyes. Orthodox ophthalmologists have undoubtedly failed to give these people the full measure of possible help, and anyone who has made a careful study along these lines and will take the time to work with patients psychologically will undoubtedly contribute much toward increasing their ocular efficiency.

Many ophthalmologists utilize orthoptics and psychological training, but most of them are unable to devote the necessary time to giving this very exacting training and are more than glad to turn it over to orthoptists or psychiatrists.

Wherein do the unorthodox orthoptists and the ophthalmologists differ? One important respect is in their concept of myopia and its treatment. To the orthodox true myopia is a condition in which the focus of the optical apparatus when at rest is anterior to the plane of the retina. To their minds no eye exercises can change this situation. The unorthodox are not interested in the optical problem as such, but in the broad idea of vision. They do not

care whether or not an image is formed anywhere.

In order to simplify the problem the subject has been investigated by the writer only as it concerns myopia, because it is thought that if the vision of the myopic patient can be shown to have been improved by exercises, the result most difficult to understand will have been accomplished.

Though it was recognized that some factors, such as ciliary spasm, could not always be excluded, it seemed worthwhile to try to collect information from as many sources as possible concerning improvement of vision based on eye exercises in myopic patients other than strabismic patients, whose poor vision is due to suppression. Accordingly about 50 ophthalmologists, a few optometrists who were reported to give muscle training, some physio-psychologists and all registered orthoptists were contacted for any information that they could give. Most of the ophthalmologists replied that they had heard about cases of improvement, but had personally encountered none. Almost all of those who had heard of cases had also heard that the improvement had been lost under stress. One ophthalmologist reported 4 cases that had been treated by somebody other than he, another reported 1 patient treated by someone other than he, another reported 7 cases of his own, and another who gives a great deal of muscle training for the purpose of improving vision reported 26 cases in detail. One registered orthoptic technician reported 6 cases; another 4; still another 4; and another 5 cases. This is a total of 57 cases. Letters were received from a doctor of philosophy who was engaged in orthoptics under medical ophthalmic supervision who stated that he had trained numerous patients who had improved greatly, many of whom earlier rejected for aviation, now were serving as flyers in our armed forces.

Though the average visual improvement of those reported was about a doubling or tripling of central acuity, a few of the more marked improvements are worth describing. A 24-year-old man had been under the care of a well-known ophthalmologist for five years. Vision without glasses had become somewhat progressively worse through these years, it being 20/200 in each eye corrected to 20/20 with about —2.00 spheres and a small plus cylinder. These tests had been made under the usual homatropine routine. This man was rejected for officer's training because of his eyes. He took muscle exercises, perhaps better described as training of visual skills, for 24 lesson periods, some as long as five hours. At the end of this time he read 20/20 each eye without correction and without squinting or any of the usual devices that help a near-sighted person to see better at a distance. The final tests were made by the ophthalmologist who had cared for him for the five years before he consulted the doctor who gave the special exercises. The refraction after the special training was essentially the same as it had been previous to the exercises, only the vision was different. In this case there was no exophoria, the effort to overcome which could have produced an accommodative spasm, nor was there any evidence of spasm. The test letters could not have been learned and the patient did not squint. A still more remarkable case is that of a patient who had 8 diopters of myopia in each eye and could see only 3/200 without glasses. After a few months' training the record shows that she could see 20/120 with each eye, the amount of myopia, however, was not greatly changed by the exercises. A third case reported by an orthoptic technician was that of improvement from 5/200 in each eye to 20/100 in each eye.

I have now had placed at my disposal the records of 57 cases in which the vision of the myopic patient has allegedly been markedly improved by one or another type of exercise. Let me stress the fact that not the same methods were used by all of those reporting. In fact, each technician has apparently used his or her own technic, and very few of those whose records I am citing were trained by the so-called Bates method; I believe only four.

Most of the doctors who are using these methods are not advocating discarding glasses, in fact they tell the patients that they will probably not be able to get along without lenses, but that they hope to improve the patient's ability to see.

First, let us consider the explanation that the psychologically interested workers offer for the results obtained. They think of the eyes as a part of the brain system of reflexes, thought patterns, sensory and motor impulses and responses. They consider every non-diseased eye capable of good vision and inhibited largely by defects not in the dioptric system, which they ignore as much as possible and about which they definitely don't wish to think, but in the subsidiary visual functions, such as background, space relationships, stereopsis, and the like. Their training consists in improving visual skills. They attempt to break up improper associations of convergence and accommodation; to relax overstimulation of the former by appropriate

prisms; to relax accommodation by the wearing of plus spheres; by rotational exercises and fusion trainings to smooth the curve of jumpy fixations. To them myopia is almost invariably functional.

These workers have little interest in anatomy. They stress the dynamic instead of the static condition of the eyes. They are interested in brain patterns. They are not much concerned whether the improved vision is associated with a lessening of myopia as measured with lenses, admitting that sometimes it is, and as often not, so associated.

Supposing that this improvement in sight occurs in some cases, is it explicable on the ground of optics? It is conceivable that learning to perceive space relations better and to improve eye movements might better one's visual concepts, but it is beyond my conception that an image can be appreciated by the brain unless it is formed on the retina. If it can be perceived otherwise, for what purpose has the elaborate dioptric system evolved through the centuries? What other incentive could there have been for its biologic development? It seems possible to me that by unremitting, purposeful training one might improve the interpretation of a blurred image, or utilize better peripheral ones.

Even such a brief investigation as I have made of this subject indicates that it demands further consideration. Possibly the theories behind the evidence are at fault and undoubtedly there are many charlatans in this field. But many others are psychologists of excellent reputation and are approaching the problem of vision along lines that are as real to them as the dioptric system is to us, and their results merit our serious study. All of the orthoptists reporting were working under the direction of ophthalmologists and their results were unpremeditated, occurring in the routine muscle training of phorias in myopic patients. The usual explanation given by the orthoptists is relaxation of muscle spasms, the cases being functional and not of organic myopia.

Whether exercises for improving myopia should be advised or not is another question. Certainly many and probably most of those attempting them will be disappointed. Much time, money, and effort will have been uselessly expended. Perhaps the improvemnt in those that do improve will be short-lived, though this has not been the experience of those reported in this group. All in all, in view of the evidence, the writer believes that the subject is worthy of further investigation.

CONRAD BERENS, M.D., New York, N. Y.: I first want to express my appreciation personally to Dr. Lancaster for having presented this subject in such a scholarly manner. It is a very serious one. Those who are charged with the duty of placing the right men in the right position in our armed forces have a difficult problem.

We know if we say that the visual acuity shall be 20/30 for the Air Forces that this may actually mean 20/40 or possibly even 20/50, because these candidates obtain training and in some way learn to see better. I do not think it is all because they meet "soft-hearted" examiners, because many of these men have been examined three or four times, and finally they return and pass.

Last year I became deeply interested in this question of visual acuity because of the great importance of visual acuity in our armed services, and suggested to the American Committee on Optics and Visual Physiology that we should have a controlled piece of research work in this country and all the members of that committee have agreed that we should.

One thing that we have never taken sufficiently into consideration is the squeezing of the eyelids. Nearly everyone believes improved vision is obtained by squeezing the eyelids and the stenopaic slit effect, but, as Dr. Post pointed out, some of these men reported that they do not squeeze their eyelids. In these cases possibly they produce a slit momentarily by blinking. This question of visual acuity may be a very serious thing. We should look into whether this is ethical and whether we should try to help the men to get in. Certainly on the color vision question, I do not believe in it. However, the Canadian Navy has permitted certain men to wear contact lenses to improve their vision. Some men say they can certainly wear contact lenses four hours and they are only on watch for four hours. I know that at least 2 men are in our flying services who wear contact lenses.

We should study the various factors concerned in this problem. I still believe that when you are finished with myopic patients they have exactly the same amount of myopia. They are put under cycloplegia before they have any training and they have the same afterward but at times some applicants do learn to see better. Certain reports I have received indicate that any improvement is merely temporary. Unfortunately retracting the eyelids seems to interfere somewhat with visual acuity even in emmetropic patients.

If I may mention the question of color vision, the armed forces, particularly the Air Forces, as well as the Navy, are examining men time after time who have been coached in the use of pseudo-isochromatic tests which are being used as screening tests here and in Canada and England. We know that the tests as given in the United States are permitting certain men to fly or perform military duties who should not be permitted to fly because they actually have seriously defective color vision. I thought that possibly after discussing this problem with some of our scientists, the type of illumination did not make a serious difference, but find that it does make a difference if we take Farnsworth's work as a criterion in this matter.

Applicants for the armed services are being trained in color vision by observing lights and by electrical treatment. Knight Dunlop, with whom we worked in the last war, claims that he is curing cases of defective color vision with 50,000 units of vitamin A. The candidates who have been "cured" by various methods and have gotten into the service, have been given the pseudo-isochromatic test, avoiding a regular sequence in presenting the plates, avoiding also viewing the plates obliquely and not permitting viewing the plates from a distance or while in movement. These men were found to have defective color vision in spite of any treatment suggested. Recently, it has been decided to select only a certain number of pseudo-isochromatic plates and that no definite sequence be used in presenting the plates. This should be an aid in excluding coached color weak individuals.

I thank Dr. Lancaster again for bringing this important subject to our attention, and I hope that we will obtain a completely scientific answer, because I think we owe it to the public and to ourselves to disseminate information based upon the results of controlled research.

FREDERICK CORDES, M.D., San Francisco, Calif.: There is one point that I feel should be emphasized in the discussion of this problem. We have heard today that in certain individuals it is possible to improve their vision by eye exercises. Under this treatment certain men previously rejected for military duty exacting normal vision have been able to pass their examination. It should be added, however, that this examination was taken under ideal conditions. These boys have been instructed to discontinue cigarettes and alcohol for several days before coming up for examination. In addition, they have all been careful to have had adequate rest.

No one has mentioned what has happened to these boys after they are in the service. We have had a little experience with these cases on the west coast which I want to mention with the idea that it may perhaps dampen the enthusiasm a little. We have had the experience that it is possible with high vitamin intake, exercise, rest, to have an individual improve his visual acuity from 20/30 to 20/20. The individual then under ideal conditions comes up for and passes his visual acuity test for the Air Corps. He usually has no difficulty in his ground work and very often finishes his preliminary flying training without difficulty. When the boy, however, is assigned to a larger ship where he has responsibility and where the nervous tension is higher he begins to get into troubel. It is under these conditions that he finds his vision begins to fail and that he has trouble flying in flying formation. Also, not infrequently there is difficulty in making a good landing and in some instances it has resulted in tearing off a wing. In addition, the realization that he is not entirely competent has a very bad psychological effect on the boy himself.

At one station on the coast where these individuals were sent for survey the doctor in charge had them put in the hospital. They were given adequate rest, mental assurance, together with the type of orthoptic exercises that they had used previous to their visual acuity examination. Under these conditions it was found that the vision would again improve. However, as soon as the boy was put back on regular duty he again had difficulty with a breakdown of his visual acuity.

Similar experiences were encountered with deck officers who in the battle zone under nervous tension found that they were having difficulty with their vision so that they were unable to see clearly, particularly when on watch where the responsibility was great.

I have discussed this problem with medical officers in both the Navy and Air Corps and they all agree that they are very strongly opposed to any attempt to improve visual acuity merely to permit a man to pass his examination, as their experience with these cases has been rather bad. As one of the officers in the Air Corps stated, this type of individual accounts for one of their major headaches. Naturally, we have excluded such things as ciliary spasm, which does not come under this group. I do feel, however, that it is well to keep in mind the fact that it may not be doing a boy a favor to encourage him

by trickery or temporary measures to pass his examination, knowing that when the time comes he may fail, and in that way, not only endanger his own life but that of the other members of his crew.

W. T. HUNT, M.D., Huntington, Pa.: To begin with, it is easily possible to believe that the retina is merely the organ for giving the trigger impulse to the visual mechanism. The rest of the visual act is, as Dr. Lancaster said, an interpretation of all our previous experience. The refractive correction which we give might be thought to be an orthoptic procedure of a kind. The functions we work on with lenses are those of accommodation and convergence, especially accommodative-convergence relationships, either positive or negative. We are trying to have the patient use a given amount of accommodation with a given amount of convergence. The glasses we give should put them into this equilibrium. The same is true of orthoptics in many phases. We are trying to develop a pattern of organization in the visual act — and I am using "visual act" in an operational sense — where there is no need for an inhibitory impulse to be sent back through the brain, an impulse to which the organism is not conditioned.

We must follow our patients with check-ups to note progress, and, of course, the checks which we use to show the relationship between accommodation and convergence include phoria, duction and blur findings, and these are dynamic findings.

Dr. Lancaster has certainly brought up a large subject.

WALTER B. LANCASTER, M.D., Boston, Mass.: I want to thank the discussers, especially Dr. Post, for adding so materially to the subject. I should like to make one suggestion. This is really a part of the subject of psychology, and it seems to me the Academy is overlooking a very important field as a course of instruction. They ought to have every year at least two courses in the psychology of vision. This subject today is only a small part of the larger field, but what little I have been able to dip into the field has been so illuminating to me that I feel sure it is worth while for the rest of you to go in and go a little farther.

THE TREATMENT OF RECENT INJURIES TO THE EYE AND ADNEXA

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OF ALL the injuries with which the surgeon in the military service has to deal, those involving the face and especially the region of the eye are the most shocking and offensive in appearance. It behooves us, therefore, in handling these injuries to bear in mind the extreme importance of caring for them in such a manner that the minimal amount of deformity will result. There are so many measures of seemingly equal significance that it may be hard to determine which one of these steps should be the first to be taken. Simple packing of the wounds may be safer than to try regional pressure or ligation. The general Army and Navy regulations advise the application of sulfanilamide, and this may be placed in the wound at the earliest possible moment. All bleeding should be stopped before anything else is done. Since a tourniquet obviously cannot be placed on the face to stop eye bleeding, we must resort to either compression and hot packs if available, or, in case of large arteries being severed or torn, to ligatures. Digital compression, if properly carried out, will always temporarily control the major part of the bleeding.

Of the utmost importance is the necessity of having all the facilities on hand to do a good job. If not, it may prove far better to move the patient farther back to the rear where these resources and advantages can be found. All bleeders should be tied with silk ligatures, cut short and left buried. It has been found that these become encapsulated and give rise to far less trouble than catgut ligatures.[11]

If the patient is in shock blood transfusion is indicated. In this respect it may be necessary to bear in mind that the patient should not be permitted to suffer unnecessary pain, since this intensifies shock. It may be necessary to administer a general anesthetic during the process of cleaning and preparing the wound. If the patient is unconscious and brain damage is suspected a neurological examination including roentgenograms should be made. In extensive injuries it may be well to wait until the patient is placed on the operating table before the tissues are manipulated to see to what extent damage has been done. Not only should the loss of any features or their tearing be determined, but they should be recorded as well, including notes as to fragmentation or detachment of bones, loss of vision, or facial paralysis. This is most important, for if these should not be recognized until after the operation there might be some question as to the actual cause of the loss of function.

After careful inspection of the wound has been made, it is thoroughly, but gently, scrubbed with soap and water, taking care to bruise or damage the tissues as little as possible. It may be necessary to apply lightly a brush where there has been a lot of dirt and grit ground into the wound. Debridement is something which has come up for a lot of discussion and some difference of opinion. It seems to be the practice of

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the majority of authorities that the cutting away of all lacerated and mashed tissues as was generally practiced during World War I has given way to more conservative methods, this trend encouraged by accumulative evidence of good end results from minimal surgical interference. We have the exposition of the two extremes in the proposal of Trueta to debride until red contractile tissue is reached as against that of Barnes'[1] simple application of sterile surgical gauze with incorporation in plaster cast. We feel that Barnes' premise is the more logical and gives the better results. Wounds must not be disturbed too often for inspection, for, as brought out by Crile,[5] this has a deleterious effect. He directs attention to the exacerbation of symptoms with each early interruption of dressings for wound inspection and comments on the advantages of watchful waiting.

Tetanus is still encountered in this war, although not nearly so frequently as twenty-five years ago. While every member of the Armed Forces is given antitetanus treatment it is not always sufficient to prevent tetanus. English observers report that in two years of fighting in North Africa, 18 cases of tetanus occurred, of which 5 had been previously immunized. The type of soil or terrain over which the fighting occurs must be taken into consideration. In North Africa, where there was little cultivation, a low rate of infected soil was found. Soil from more fertile and cultivated stretches was found to contain as much as four to five times the infective rate of the desert. Tetanus toxoid should always be administered where the wound is deep and especially if the opening is small, dosage being usually 3000 units of antitoxin. The English believe that the quantity of toxoid now being administered for preliminary immunization is not adequate and that three injections instead of two should be given.[2]

Because of the presence of swelling and the possible danger of meningitis, some surgeons make no attempt to reduce facial fractures involving the orbit or the immediate surroundings of the eye. Hook[6] thinks that the principle of early reduction of the fracture is as important in wounds of the face as elsewhere in the body, that cutting away of any considerable amount of soft tissue is never permissible and that early cleansing and suturing without tension is extremely important. Small fragments of bone will ordinarily be removed.

Dirt and foreign bodies in the wound should be carefully removed. This is especially true in the case of dynamite explosions where small fragments of clay and clothing may be blown into the wound. In this respect it may be of value, as stated before, to know something of the character of the soil where the patient was wounded. Gas gangrene infections of the face are rare, but, if feared, antitetanus and gas gangrene serum may be used at the same time.

While there is no question as to the advantage of using the sulfonamide compounds both locally and internally, it must be borne in mind that they cannot be used without taking certain precautions. It has been shown[7] that necrotic material or pus contains sulfonamide inhibitors; hence every effort should be made to eliminate these substances before sulfonamide therapy is started. Again, implanted sulfonamide compounds may cause an exudation of tissue fluids into the wound and sloughing of the wound edges has been noticed where the wound has been too tightly closed. In war wounds, as shown by Long,[10] experience has demonstrated that primary closure is dangerous, and with few exceptions, the wound should be left open. Because of the vascularity of that area face wounds heal better than wounds in other parts of the body. Adequate use of implanted sulfanila-

mide into the wounds of our casualties at Hawaii prevented wound infection, thereby permitting debridement of some wounds many hours after they had occurred. After a few days the wounds were so clean that some were secondarily sutured, and healing by first intention took place. Long also states that out of the welter of conflicting experimental and clinical reports, one fact seems to emerge, namely, that while the logical implantation of sulfanilamide may interfere to a certain degree with wound healing it appears to be the least harmful of the commonly used sulfonamides to regenerating tissues. In wounds that are closed by primary suturing the amount of crystalline sulfanilamide implanted should approximate 0.05 Gm. for each square inch of wound surface. In contaminated wounds in which closure is not indicated, or infected wounds, 0.1 Gm. of crystalline sulfanilamide per square inch of wound surface should be used. Not more than 10 Gm. of sulfanilamide should be given locally to any one patient within a twenty-four hour period.

For adult civilian injuries, according to Long, the initial peroral dose of sulfadiazine should be 2 Gm. to 4 Gm. given either immediately before or after definitive treatment. This should be followed by 1 Gm. of the drug every six hours for two days, and following this, doses of 0.5 Gm. should be given every six hours for eight days. For adults wounded as the result of enemy action the initial dose should be 4 Gm. given as soon as possible. After this no more of the drug should be given until after definitive surgical treatment has been completed. Then 1.0 Gm. should be given every six hours, day and night for seven to ten days. It is important to maintain a daily output of the urine of at least 1000 cc. during the period of treatment in order to lessen the possibility of the occurrence of renal complications.

The experience of Cope[4] in the treatment of 39 patients brought to the emergency ward of the Massachusetts General Hospital from the Coconut Grove night club fire emphasizes the importance of the better care of early shock, which was instituted immediately in all of these cases. Intravenous administration of plasma was given as soon as possible, in many cases while the patient was still on the stretcher. No debridement or cleansing was done, merely the application of sterile boric acid strips secured in place by pressure bandages. The The dressings were not changed until the fifth or tenth day. Before removing the canula or needle already in place for the plasma transfusion, 2 Gm. of sulfadiazine were administered. Cultures of fluid twenty-four hours to fourteen days old were taken under sterile conditions from the blebs. Examination by Lyons[8] revealed in only a few instances nonpathogenic saprophytes of normal skin. The treatment appeared to confine the bacterial infections which occurred in all of the deep burns to the devitalized tissues and to protect the living cells from invasive infection or pronounced local necrosis.

The principles of treatment of war injuries discussed by Dr. Converse[3] last year are applicable. I refer mainly to the early closure of wounds. When that is not possible where bulk of devitalized tissue must be removed and the wound cannot be easily brought together, skin graft at the earliest possible moment is necessary. Where chemotherapy has been instituted, the chances are that a skin graft will be successful. Granulation tissue is removed since it results in the formation of fibrous tissue and becomes infiltrated with fibrogen, resulting in scar tissue. There is practically no sensation in granulation tissue, so that it can be easily removed without the use of an anesthetic. As soon as the bleeding has stopped and there are no

blood clots or strands left on the surface, a thin layer of sulfanilamide is dusted on the surface, over which the skin graft is placed. In a series of 110 cases reported before the Royal Society of Medicine last year 88 per cent of this type of graft was completely successful.

Dr. Converse related that there were many cases of death from explosion of bombs where there was no external evidence of injury to the individual. We find this also in injuries of and around the eye. During World War I we found many cases of rupture of the choroid without even a bruise of the external eye or lids. We find them now also. One patient from the South Pacific presented a very pronounced exophthalmos of the left eye following an explosion, without any external cut or bruise. A marked bruit was heard over the eye and forehead. The conjunctival and retinal vessels were dilated on that side. The central vision and fields were normal. Ligation of the internal carotid artery on that side by the attending neuro-surgeon at the Naval Hospital has resulted in the almost complete recession of the protruding eye. A preliminary exploratory craniotomy had confirmed the diagnosis of aneurism of the lateral sinus.

Through and through cuts of the lid involving the margin are handled so that no loss of the marginal conjunctiva occurs. This margin is carefully trimmed on either side and an eliptical piece of the tarsus excised on each side. The lid margins are then brought together so that the conjunctival edges are perfectly approximated, by means of a deep bite taken vertically through the conjunctival margin, emerging on the cut edge of the tarsus. A second bite is taken on the opposite side in reverse order. When this is tied, if the lid edge is not perfectly in line, the stitch should be removed and reapplied. A second stitch through the tarsus in the middle of the cut will bring it together and prevent "dimpling in" of the lid margin.

Where the cuts in or around the lid are deep, deep sutures should be brought together first before tying the skin. The outstanding point to be remembered is that accurate approximation of the wound at the time of first aid, or very soon thereafter before swelling or edema distorts the relationships, may save months of later hospitalization with possible repeated repair operations and untold pain and mental suffering. In case of severe tearing and destruction of tissue, unquestionably destroyed tissue must be removed and the wound then cleansed with as little irritation as possible. However, it may be desirable to retain torn fragments of undestroyed skin, even though very small, for they may be of great value in the healing process of the wound. Sometimes these skin fragments are so tangled and distorted that it requires time and painstaking effort to disentangle them and put them into their proper places.

Immediate repair may involve the same principles of plastic restoration as if there had been loss of tissue, especially that of skin, in addition to mere cutting and tearing. The problem then resolves itself into the same situation as that in which a small or large bulk of tissue has been removed in cases of malignancy. I would advise all eye surgeons to do nothing in these cases except cleanse carefully, institute sulfanilamide or other prophylactic measures and send the patient back to the rear, or even to a base hospital in the States, unless the surgeon is well experienced in this sort of repair. More permanent damage may be done by improper repair than by waiting until an experienced man can be reached. While it is my firm conviction that, other things being equal, the eye surgeon can do much better work around the eye than the general plastic surgeon, when they are not equal and the eye sur-

geon has not had this special sort of training, the general plastic surgeon can do it better. It is true that plastic surgery has some definite, well established principles, but on the other hand, each case presents a distinct and separate problem which may possibly have to be solved as the surgeon proceeds. The mere fact that an eye surgeon is proficient in the ordinary run of eye operations does not imply that he is able to do complicated plastic repair work, but if he has had experience in this line the eye surgeon can handle the delicate tissues around the eye and orbit much better.

In case of perforating injury to the eye one always suspects the entrance of one or more foreign bodies into the globe. Roentgenograms will determine this. If possible, it is well to obtain some of the material similar to that which penetrated the eye and lay it on the edge of the film. If it casts a shadow we would expect it to do so in the eye. This procedure is particularly valuable in the case of glass, of which many forms do not cast a shadow at all.

In the cases of presence of intraocular foreign bodies seen in the service of the Navy very few respond to the magnet. Not one has been seen by us in any of the Coast Navy Hospitals except those not contracted in actual combat. These foreign bodies are best removed with a powerful magnet through the sclera. The cut in the sclera should be made with a very sharp cataract knife as near as possible to the localized point and must be considerably larger than the greatest estimated diameter of the foreign body to be removed. It is well to train the patient for what is coming before turning on the current and have him approach the tip of the magnet, with the operator holding back his forehead so that he will be assured that it will not bump hard against the tip. The current is turned on after the tip has entered the wound. Often it is necessary to turn the current off and on several times before the fragment is dislodged. If one eye is gone and the other eye has multiple foreign bodies, it may be wise not to make an effort to remove any of them unless some are near the surface and can obviously be removed without exerting too much trauma. If both eyes contain multiple foreign bodies, as sometimes happens from land mine explosions, and one of them retains light perception while the other does not, I would advise immediate removal of the blind one to eliminate the possibility of sympathetic ophthalmia rather than retain this useless globe for mere sentimental reasons.

It is astounding what a tremendous amount of damage can be done to an eye and have it recover with some vision, even though it be slight. I can cite one case where the right eye was torn out by an explosion of a hand grenade and the left eye filled with foreign bodies. Light perception was fair and projection limited to the lower field. We treated this case expectantly. His perception and projection gradually increased after a few months. At the time of his discharge from the hospital he was able to count fingers in a small field below at a distance of one foot. No details of the fundus could be seen in this case on account of vitreous bands and opacities.

An officer was flown back to the Navy Hospital in San Diego in three days, following the first Guadalcanal battle. His body was full of fragments of metal, one large one being removed from the knee joint with complete recovery of function. He had a glistening foreign body in the left lens just above the center and behind the anterior capsule. It looked like aluminum and was non-magnetic. One could easily trace its path through the eye by the scar in the cornea, the hole in the iris and in the anterior capsule of the lens. This case was

also treated conservatively. Six months after the injury had been sustained his vision was 20/20 in that eye. It never did produce any redness or give him any pain. He is now back on duty.

Where tears are through the sclera, conjunctival sutures will bring the wound together. Injuries to the ciliary region are always potential sources of the development of sympathetic ophthalmia and must be carefully handled and watched. The anterior chamber of the other eye must be carefully searched for cells, even though the vision is not disturbed and the eye is without visible signs of inflammation. At the first sign of pitting of the injured eye, which means eventual shrinking of the eye, it is much wiser to enucleate, even though the eye still retains some amount of vision. It is well to explain to the patient that it is far better to have one seeing eye than two blind ones. Conjunctival tears usually require no sewing unless they are quite extensive. Scleral ruptures are not infrequent, sometimes coming from explosions without any surface manifestation. They practically always mean loss of the eye. I can recall 1 case where a large rupture of the sclera was sewed together with conjunctival sutures and in which recovery of some useful vision occurred. Such cases are exceptions. Rupture of the choroid sustained in the same manner is frequently encountered. I know nothing that can be done for this. Detachment of the retina due to traumatism is one of the most adaptable types for a favorable prognosis with electrocoagulation.

We have seen 1 case of penetrating injury which resulted in the formation of an iris cyst in the anterior chamber. The cyst, including the contained foreign body, was successfully removed. Foreign bodies of the cornea are, of course, an everyday occurrence. Multiple foreign bodies in the cornea are handled according to their character, depth, multiplicity and the degree of irritation they cause. It is not only sometimes unnecessary to remove powder burns, it is not always advisable. If they do not obstruct the pupillary area and are causing no irritation, more harm can result from their attempted removal by increasing the scar than by leaving them alone. Each case must be considered as a separate problem of its own and treated accordingly. If the cornea is cut through and the iris protrudes, the procedure is to flush out the conjunctival sac, then grasp the iris firmly with an iris forceps and pull it out clear of the wound so that it is freed from its attachment. Cut close to the cornea and gently massage the wound until the iris has sunk back into the chamber and no longer adheres to the cornea. This may be done only if the patient is seen within a few hours after the injury, for the iris becomes rapidly adherent to the wound. If the iris cannot be pulled away from the wound, then, with a very sharp pointed iris scissors, cut the protruding iris as close to the corneal surface as possible. Sometimes, in an apparently clean wound seen shortly after the injury one is tempted to try replacement of the prolapsed iris because it may preserve the pupil and give a better cosmetic result. This is very risky and I would not advise the attempt. In the first place, we have no assurance that it will not prolapse again with the first outgush of aqueous. The main reason is the danger of pushing contaminated material into what may have been a perfectly clean chamber. It is seldom necessary, or even advisable to sew the cornea. If the cut is extensive and runs clear across the cornea, then an anchoring stitch through the superficial edges of the cornea at one end will almost always suffice. One must be sure that the stitches put in the opposing sides are corresponding points. Fortunately we have the great advantage of the use of

the sulfa drugs, given both locally and perorally, to thwart later infection.

In perforating injuries involving the lens, it may be best to wait until all irritation is gone before attempting to remove the lens, unless the swelling has been very rapid and the wound small so that the tension is increased and the patient is suffering pain with severe local reaction. Adrenalin chloride, 1-1000, will nearly always control the tension in this type of swelling. If not, opening of the wound and washing out the lens remnants may save time and forestall a great deal of suffering. Dislocation of the lens nearly always results in increased tension and subsequent loss of vision. It is therefore advisable to remove the dislocated lens as soon as the congestion has disappeared, or it may be necessary to do this sooner. It can be done by the Smith method with little or no loss of vitreous, even though the vitreous be fluid. Tension should be reduced before operation if possible. In fact, it may be wise to give a weak solution of pilocarpine in these cases to avoid just this.

Burns of the conjunctiva and cornea due to alkali or acid should be treated by prompt flushing of the conjunctival sac with water and with considerable force. This not only will mechanically get rid of all the harmful material, but also neutralize by dilution. If the case is seen early enough no other treatment may be necessary. If the burn is superficial, cool compresses applied for about fifteen minutes at a time over the closed lids relieve the pain and photophobia and do no harm. Where the burn is deep, as from lye and other severe escharotics, warm applications are indicated to increase the vascularity so that the cornea will get sufficient nourishment. Cold is therefore contraindicated. Atropine is used in all cases where there is ciliary injection and for as long as this persists. Glaucoma can occur in injuries, even in very young people, so that the tension must be watched whenever atropine is used. Yes! and even when it is not used. The severity of the burn damage cannot always be judged by merely watching the cornea. In lye burns I have seen the cornea remain clear for days after the injury and then suddenly drop out within twenty-four hours or less. The index is the color of the conjunctiva. As long as the conjunctiva and the underlying sclera remain red one need not fear any more severe damage to the cornea than already manifests itself. It is when the eye looks too bleached and examination reveals that the vessels of the conjunctiva and underlying sclera have been destroyed that we need fear total loss of the cornea. That is the reason that cold is contraindicated in deep burns and warm applications are the thing to use. If the pain is too severe it may be relieved by instillation of 0.5 per cent pontocaine. However, even pontocaine and butyn soften the corneal epithelium and delay healing, so we don't like to use them unless absolutely necessary. Cocaine should never be used. Regular separation of the lids may possibly prevent adhesion of the lid to the eyeball; but eventually, if much tissue has been destroyed symblepharon will occur, regardless. Some authorities feel that Denig's recommendation to place a mucous graft immediately is good practice. I have had no personal experience with this method.

Pickrell's[9] solution of 3 per cent sulfadiazine in 8 per cent trienthanolamine sprayed over the surface of a burn of the face or lids seems to be the best immediate treatment thus far used. It has a pH of 8.3, is clear, odorless and does not stain, and no previous traumatizing scrubbing is necesary. It makes the patient comfortable, forming a tough, thin, transparent and pliable eschar. It is so elastic that it does not interfere with mo-

tion of the lids. Sulfadiazine jelly may also be used in the eye.

Injuries to the eye from gas have not been encountered so far in this war, but we must be prepared for them. Burns from tear gas are usually merely irritating and need only paliative measures, unless the gas happens to be splashed into the eyes, as can happen when it is fired at very close range. Its burn then resembles that of a strong acid and is treated as such. Severe injury from mustard gas may be produced from as low a concentration as 1-10,000,000. The clinical and pathological effects of mustard gas are similar to those of an alkali burn. In a large percentage of cases there is only a very mild irritation of the conjunctiva which subsides within a week or two. In a still smaller number the conjunctiva is severely burned with a white, chemotic band between the lids and a dotted roughening of the cornea. Recovery requires five or six weeks in this degree of burn. Where the cornea is deeply involved with erosion, the healing is very slow and stubborn, with frequent relapses. It may require several months before healing is complete. The symptoms from mustard gas vapor are not present immediately but make themselves manifest after a period of from a couple of hours to as long as forty-eight hours, dependent on the length of time exposed and on the concentration of the gas. First aid consists of irrigation for several minutes with any bland, non-irritating solution, using atropine if the cornea is involved and also sulfathiazole jelly or ointment to help prevent secondary infection. Burns from Lewisite gas produce more rapid destruction of the tissues and require immediate and abundant flushing with any watery solution which will dilute its effect. One per cent or 2 per cent bicarbonate of soda solution is advised if available. Phosgene gas causes very little local irritation, although we may look for retinal hemorrhages secondary to edema of the lungs if that occurs. Glycerine instillations are often recommended; but glycerine itself produces irritation and for that reason is not advised.

In summing up, let me emphasize the fact that in the treatment of war injuries to the eye and adnexa the same general principles of medicine and surgery hold good here just as in any civilian case. There is no general hard and fast rule, and good surgery is not the index of any one single method. After all, the final outcome depends on the personal equation of the one in charge, who must have the ability to read the wound and be able to prophesy its future. He must know when as well as how to cut, how far to cut and when he has done enough. In war wounds he can close a higher percentage of contaminated wounds by primary suture than he can with antiseptics, by painstakingly removing irreparably damaged tissue with such accuracy that that natural defenses of the revised wound become its best protection against contagious matter. In the last analysis, the wounded soldier or sailor depends on the sane and sound judgment of the attending doctor.

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DISCUSSION

WENDELL L. HUGHES, M.D., Hempstead, N. Y.: Dr. Wiener has discussed and emphasized many points in which I am in hearty accord. He has been unusually fortunate in being able to study some of the cases wounded in the present war, which has not been my lot. My discussion must of necessity be limited to the consideration of general principles.

These are a few points about which I would like to speak —

1. Of primary importance in any wounds in this region, is the preservation of a seeing eye. It is remarkable how much damage may occasionally be suffered by the lids with practically an intact globe. Frequently the early management of these cases is of extreme importance and will decide whether a clear cornea or a scarred cornea will result or, indeed, whether or not the eye will be saved. In extensive lacerations and tearing of the lids with loss of tissue it is important to keep the cornea protected at all times. This is, of course, best done by covering it with conjunctiva if at all possible. Even if there is only a small fragment of conjunctiva left this should be arranged to cover the cornea by the emergency surgeon first seeing the case. If necessary the lids can be sewn together so that no skin, lashes or other rough structure will be able to rub against the cornea. The patient is then sent for special plastic care where the eye plastic surgeon will be able to care for the injured patient as soon as possible. Sulfanilamide in the wounds combined with a moderate pressure dressing will keep swelling and inflammatory reaction at a minimum and enable better replacement and repair of tissues to be done at an early date. Internally, sulfadiazine is of great help in keeping reaction due to infection at a minimum.

It is, of course, not advisable for the emergency surgeon to try any extensive repair work or plastic work unless or even if he has had special training in this field. The patient should be sent as soon as possible to an eye surgeon specially trained in plastic work or to a plastic surgeon who has had some special eye training. The special structure, delicacy and importance of the structures with which one is dealing cannot be overemphasized.

2. In regard to foreign bodies in the eye or orbit, unless the foreign body is visible a roentgenogram is of prime importance, especially if one is able to obtain the services of a man who is specially trained in localization of foreign bodies. Many aluminum and glass foreign bodies may be visualized, if located in the anterior segment of the eye by means of a bone-free technic taking the picture from the temporal side with the corner of a dental film (in duplicate) placed at the medial canthus. These foreign bodies will not show in the usual anteroposterior picture unless unusually large or containing other metals as alloys. Foreign bodies outside the eyeball unless large or definitely producing trouble are best left alone. Of great aid at the operating table in this case would be the Berman foreign body localizing instrument. Since it is frequently extremely difficult to find foreign bodies in the soft tissues the use of the foreign body probe can be of inestimable value. A piece the size of a pin head, if it is of steel or iron, will give a good reaction on the dial of the instrument if the end of the probe can be passed to within $\frac{3}{8}$ of an inch of it. Much less reaction is obtained from copper or brass and no reaction from glass. Its field of greatest usefulness is in foreign bodies in the orbit or those lodged firmly in the sclera. The exact position over which to make the incision in the sclera can be easily determined if it is within reach of the instrument.

In the case of metallic foreign bodies within the globe this instrument may be of occasional value if the foreign body is of iron or steel. However, these foreign bodies give little difficulty to the eye surgeon who has available the proper equipment to handle such cases.

Glass, aluminum and lead produce little inflammation from their presence in the eye. If a small copper or brass foreign body is

contained within a clear lens, the capsular rent having healed, it may sometimes be retained with very little reaction. Elsewhere within the globe these metals usually produce severe reaction leading to loss of the eye unless the foreign body can be extracted. If the lens or vitreous is clear one can occasionally reach into the globe through a scleral opening, under ophthalmoscopic guidance, and grasp a foreign body with a duckbill forceps.

Personally, I prefer the anterior route for the removal of most magnetic intraocular foreign bodies within the camera posterior of the eye. The technic of the use of the magnet is somewhat different in this procedure than that described by Dr. Wiener. I usually try the small hand magnet first and if this does not bring the foreign body anteriorly, the giant magnet is used. The current is applied intermittently first at a considerable distance from the eye, gradually bringing the tip closer to the eye until the patient experiences pain or until a visible movement of the iris or sclera is discernible on applying the current. The foreign body is drawn toward the sclera just behind the ciliary body and then the eye is rotated so that the magnet will pull the foreign body up around the lens into the posterior chamber. The small hand magnet then suffices to draw it parallel to the anterior surface of the lens through the pupil and through a small keratome incision in the cornea temporally. A small bubble of air inserted into the anterior chamber will prevent any tendency for the iris to become adherent in this location if the patient's eye is held so the air bubble floats up into that portion of the anterior chamber angle. In two to five days the air bubble is spontaneously absorbed.

Dr. Wiener did not mention whether or not he uses any diathermy current around the site of the foreign body extraction when done through the sclera. It has been my custom to use this modality around the site of the scleral wound after tying the closing suture. I would like to ask if he has been in the habit of following this procedure. If we accept a tear in the retina as a factor in the production of a detachment of the retina it would seem that this is a logical procedure.

I wish to thank Dr. Wiener for the opportunity of studying and discussing this most interesting paper.

HAROLD F. WHALMAN, M.D., Los Angeles, Calif. With regard to the removal of intraocular foreign bodies by way of the posterior route, I wish that Dr. Wiener would discuss the advisability of using diathermy around the operative wound to prevent retinal separation and also the advisability of hospitalization of these cases for the same reason. I think that this is a very important point and may entail an added medico-legal aspect to the removal of foreign bodies. I should like to hear these points discussed.

R. J. BILLINGS, M.D., Pittsburgh, Pa.: Slow absorbing or unabsorbable intraocular hemorrhages lead to fibroplastic infiltration and later fibrous bands and adhensions, resulting in secondary glaucoma, detached retina, and other complications. Iontophoresis with acetylcholine and eserine hastens the absorption of these hemorrhages and exudates.

MEYER WIENER, M.D., Coronado, Calif.: First of all about the covering of the cornea; I do not think that is necessary. I have never practiced covering the cornea with conjunctiva. In corneal wounds I do not use any conjunctiva flaps. I know that is contrary to the general useage at present. I know especially that Dr. Gifford believes in it very strongly. I have seen some injuries that came in with a corneal wound through and through, maybe half of the cornea, and I never saw them until they were healed and they had no infections, because there is constant draining out of the aqueous and, probably, all of the bacteria as well.

As to the use of sulfa drugs, that is discussed in my paper, so I won't say anything more about that.

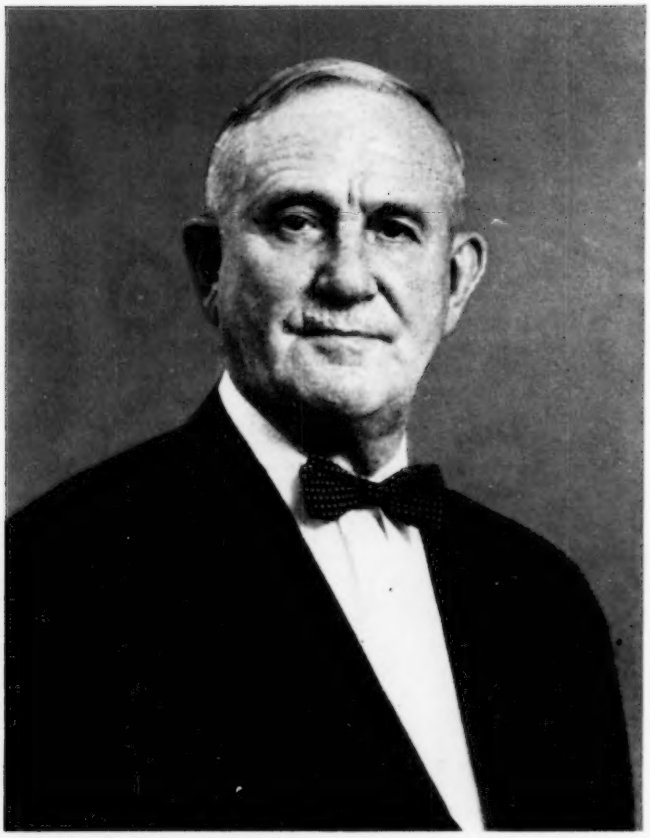
These cases should be handled by the plastic surgeon or by the eye surgeon. I feel very keenly about this. I feel that wherever a man in ophthalmology has had experience in plastic surgery, he is a whole lot better than any general plastic surgeon. I have seen the results of the very best plastic surgeons. I am not casting any aspersions on any plastic surgeon, but if a man has had experience in ophthalmic surgery plus the proper experience in plastic surgery of the eye, I am sure he does much finer, much neater, and much better work than the general plastic surgeon. However, there are very few real eye surgeons — I am not saying this so that you will send me patients because, as you know, I am out of practice. I haven't practiced for more than seven years, so it is not at all personal. Whenever the eye surgeon has not had that experience, then the general plastic surgeon does very much better. There is even a much greater difference there. I have seen one horrible experience so far, in this war, where a very well trained eye doctor,

a young doctor from one of the best institutions in the country, got the eye surgeons in very great disrepute in the Navy because of the manner in which he tried to make a plastic repair; and it was finally sent to the plastic surgeon, who got a nice result, holding it up as an example to us.

As far as using the magnet is concerned, I was taught to use the anterior route. I saw Haab's magnet when he first developed it in Zurich. The only large, giant magnet in this country at that time was at the New York Eye and Ear Infirmary. I myself constructed a still larger and still more powerful one in a machine shop in the Manual Training School in St. Louis. It was powered with a 500-volt current, and the longer I worked the more I was convinced that the posterior route was best if one had good localization.

I have not practiced diathermy for the reason that when I first started in ophthalmology I did a great deal of industrial work. After a few years I did very little, but we had a lot of perforating injuries and we had a lot of foreign bodies. We took them out by the posterior route. I have never seen one case of detachment of the retina following use of the big magnet through the posterior route. I don't think you can compare that in answer to Dr. Whalman, with the ordinary detachment, because in ordinary detachment of the retina we have something more. We have a diseased choroid to deal with, and we assume that we have a normal choroid and retina when we have a perforating wound from a clean knife wound in extracting a foreign body.

Guest of Honor



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Editorials

The Convention

The annual conventions of the Academy, which have been held without interruption since 1896, have grown in leaps and bounds since the last war. The activities of the Academy during the week of the convention have been augmented by activities of affiliated and component groups having special interests until five days are now required to complete the Academy program. More scientific papers and discussions are now available at the convention than ever before in the history of the organization and the scientific and technical exhibits are equal in quality to those of any national medical organization.

Last year the attendance reached almost two thousand and the facilities of the hotel were taxed. This year the anticipated attendance is more than two thousand and the hotel situation is but little changed. Man power is limited not only by procurement but distinctly limited by government order. That is not the fault of the hotel management. During the heaviest occupancy in history the management at the Palmer House has made it possible for the Academy to hold its annual meeting in a midwest city with the best possible transportation facilities and has heartily thrown open every possible hotel facility permissible under war-time restrictions. Members of the Academy will bear in mind that the purpose of the convention is to aid in every way dissemination of knowledge and information concerning the recent advances in our medical specialties. Even under adverse travel and housing conditions the objectives of the Academy can be achieved. Inconveniences in travel and housing are a result of war-time conditions and cannot be permitted in any way to interfere with the success of the meeting.

Members attending the convention in October will bear in mind that those are

best served who make their wants known and whose wants are not unreasonable. Railroad transportation to and from Chicago should be obtained well in advance of the meeting. Reservations at the hotel should be made at the earliest possible moment. By government order hotel rooms are presumed to be vacated by 3 o'clock in the afternoon instead of 6 o'clock as has previously been the custom. Reservations made and promised for a certain day are not available until after 3 o'clock of that day. Members asking for reservations should bear this in mind and inform the hotel management in writing for reservations the exact time of their expected arrival.

Members who have been accustomed to room service are reminded that through restrictions ordered by the government, personnel for room service is markedly reduced. Members should not expect to have meals served in their rooms and maids available on call at all hours of the day and night. One should bear in mind that the real purpose in coming to the convention is to participate in an educational program that has not been restricted by any government necessity, while the housing facilities will be made the best possible under existing conditions.

Home Study

The educational objectives of a large organization of medical specialists cannot be lowered in time of war. The truth of this statement is self-evident, for during the longest period of duress in the history of our country the eager interest in medical education on the part of teachers and students has steadily increased and is growing with every passing year.

The Academy has developed educational facilities unique among medical organizations. For more than five years it has carried on a year-round program of home study that has now become so popular that only a fraction of the applications received for the courses can be accepted. These applications come from three large groups, namely, men in military service, residents and interns, and specialists in civil practice who wish to utilize the machinery set up by the Academy through its home study and lecture courses to bring their clinical practice up to date. These applications are mute testimony to the keen desire of ophthalmologists and otolaryngologists to keep in the forefront of medical advances. The faculty is an organization of medical teachers and students in ophthalmology and otolaryngology and in plastic surgery of the head, face and neck, whose sole purpose, so far as this organization is concerned, is to participate in educational activities by means of lectures, demonstrations, motion pictures and forums for discussion of scientific phases of clinical practice. Their interest is maintained the year round but reaches a climax during the convention held once each year.

Jackson Memorial Lecture

It is fitting that those who have accomplished much for the welfare of mankind should be remembered through suitable memorials in recognition of the good that they have done. How best to honor those ophthalmologists who have so distinguished themselves is difficult to decide. The most that can be done is to establish a memorial that will call to the attention of physicians in the future the life and writings of such a man and express the love and esteem in which he was held by his contemporaries. Many of those who were devoted to Dr. Edward Jackson and recognize him as the leading figure in ophthalmology in America and one who pointed the way in many phases of medicine beyond his chosen field have given much thought to

this. On his seventieth birthday a memorial volume was compiled by his friends and the occasion was celebrated by a testimonial banquet in Denver, his home for the last fifty years of his life. Another such volume would be superfluous and it was thought that a compilation of his publications alone would not be a sufficiently living reminder of him. A history of ophthalmology in America based on his life as the central thread was another idea favored by some. Its difficulty of accomplishment prevented this undertaking for the time being at least. Finally agreed on was the present plan of a lecture sponsored by the American Journal of Ophthalmology to be given under the auspices of the American Academy of Ophthalmology and Otolaryngology annually, to be known as the Jackson Memorial Lecture. Because of his special interest in the instruction of young men and his lifelong endeavor to inculcate in them an interest in research, it seemed appropriate that this lecture should be presented at a meeting to which all new members were particularly invited.

The Business Meeting of the Academy for many years has been poorly attended. The membership seemingly has thought that the Academy has been conducted by a group who have been especially interested in its efficient administration and who have done such splendid work in planning for and conducting the organization that their own efforts were unnecessary and that merely to give approval of the administration was insufficient reason for attending a somewhat dull meeting. However, non-attendance at this meeting, the only executive session at the Academy for all members, is scarcely fair to those who give so generously of their time to make the Academy the success that it is today by planning multiple programs for the pleasure and instruction of all its membership. There are many committees that report at this meeting the work of the year, which often has been arduous and time-consuming. It is no more than right that the membership should turn out well to express, by their presence, appreciation for the efforts of their fellow members.

It is particularly important that new members, including all who have been in the association for only a few years, should attend this meeting which reflects so directly the work of the Academy, the machinery that keeps the structure moving. It is the idea of the Council that by having the Jackson Memorial Lecture given at this time and by selecting subjects particularly appropriate to the young man, and by especially inviting new members to be present, the meeting may be more attractive and a better attendance assured. We have been fortunate in securing as the first speaker Dr. William H. Crisp of Denver, Colorado, for many years one of Dr. Jackson's most intimate friends and well conversant with Dr. Jackson's ideas, one who has been an editor of the American Journal of Ophthalmology for many years and who succeeded Dr. Jackson as Editor-in-Chief. Dr. Crisp has been a man especially interested in the education of ophthalmologists, one time member of the American Board of Ophthalmology, and an outstanding writer of editorials on ophthalmic subjects.

The President of the Academy issues a most cordial invitation to all members of the Academy and guests and especially to the neophytes to attend the Executive Session and to listen to this first Jackson Memorial Lecture which cannot fail to be of interest to them. Its application will not be confined to ophthalmology alone, but will be of importance to all who are concerned with medical education.

LAWRENCE T. POST, M.D.
President

Graduate Medical Education with Special Reference to Plans for Postwar Requirements

IN ANY postwar plan for continuing education, one must refer to that program of graduate study which calls for a prolonged period of instruction in an educational institution after the internship has been served. It implies concentration in some particular field, research perhaps into the reasons for certain methods of practice, and may lead to the Master's degree for those who have satisfactorily completed prescribed requirements. It is the highly approved and accepted method of acquiring the knowledge and technical skills of the specialties and the program for which the vast majority of graduates of medicine invariably strive. Unhappily, in times of peace it is not open to all who desire specialized training and obviously in this present emergency it can be offered in its consummate fulfillment only to physically qualified male graduate students, and women.

To be specific, this curriculum of continuing education which is the outstanding feature of American medical education today, calls for a rotation service which creates a staff ranging from the resident at the bottom of the class to the oldest individual in years of training who is approaching the termination of a three, four, or even five year period of study. In this group there are staff members of varying degrees of experience and skill who transmit their knowledge of the subject to those whom they outrank and receive instruction in turn from the superior officers on the staff. Thus each member of the group assumes the function of an instructor and the responsibilities thus imposed upon him are invariably followed by a diligent search for knowledge and information which he may pass on to those whom he is expected to serve.

The maintenance of a rotation service affords an opportunity for staff members to work shoulder to shoulder each day, to give their knowledge, experience and technical instruction to their subordinates, and to receive the compensatory benefits of superior training and judgment from their elder colleagues. Groups of young men on rotation services of this type are best served when carefully supervised by senior physicians in the department, who have had years of experience in teaching and the practice of medicine.

This plan appears to be one to which we may turn for the rehabilitation of graduate medical education when the war has come to an end. We must hold it intact even though in a limited form during this emergency, so that our programs of continuing education will not pass into a period of decadence from which recovery may be unduly prolonged. If we retrogress, we may suffer the risk of having medical education, and perhaps, too, medical service taken out of our hands and placed in those of hierarchies.

Of extreme importance now is the mobilization of all our resources by those of us who are not actively engaged in military service, in order that we may not only reconstruct, but greatly expand our programs of medical education immediately after the war. Any medical school that admits a responsibility in grdauate education must face this obligation with a clarity of objective and a precision of action, if it is to meet the needs of medical officers who will return to peacetime practice.

Consider, if you will, the vast group of medical men now serving with the Armed Forces, who some day will return to our educational institutions for the consideration and help they so richly deserve. Some of them will find opportunities for continuing education in line of duty, but the number thus favored

will not be large. Only a selected few will be privileged to review the basic sciences while in military service. Consequently there will be a great need for the establishment of a postwar program of continuing education in these fields which are so indispensable to the creditable practice of medicine.

Therefore, let us give serious thought to this problem, that we shall not be remiss in the preparation of broad programs of graduate study that will meet the requirements of physicians who will some day return to our institutions for the service which it is rightful that we should render. It is obvious that the medical schools of this country and their affiliated hospitals have neither the facilities nor the faculty personnel to conduct the comprehensive schedules that the exigencies will demand. But it is entirely conceivable and moreover, practicable, that the medical schools might consummate an affiliation with hospitals within the state in which they are located, that are equipped to offer graduate training, and thus organize programs of large scope that will adequately meet the needs of both our medical officers and civilian practitioners of medicine. In this close relationship, the medical schools will assist in the organization of curricula, library facilities, the maintenance of educational standards, and furnish instruction in the fundamental sciences of medicine. Experience of the past few years in our own institution indicates that residents in the hospitals of southern Michigan find little difficulty in traveling to our medical schools once a week for a day's instruction in one or two of the basic sciences. The resident engaged in clinical work in the hospital five days out of the week thus finds inspiration and guidance for the energetic pursuit of fundamental subjects on his sixth day in the laboratories of the medical school. The requirements of continuing education today and those of greater magnitude for the future challenge us to a plan of action which jointly and cooperatively embraces the maximum utilization of all educational facilities within our state.

OPPORTUNITIES FOR GRADUATE MEDICAL EDUCATION

1. It is the general consensus of opinion that adequate facilities for graduate medical education are not available at the present time. Even junior rotating internship services are poorly organized and suffer from the lack of instruction in many hospitals in all parts of the country.

2. Statistical studies by various agencies in organized medicine clearly indicate that hospitals offering training in the specialties are too few to meet the demands of candidates. Nevertheless, the various specialty boards are imposing requirements for certification which can be met only by the comparatively few individuals who are favored by appointments to the limited service now in existence.

3. It is generally agreed that the best form of graduate medical education is offered by accredited hospitals which are affiliated with medical schools.

RECOMMENDATIONS

1. An affiliation between the medical schools and the hospitals within the respective states which meet educational standards acceptable to the medical school.

2. The qualifications of affiliated hospitals must cover adequate facilities, equipment and personnel. Each chairman of a department of the medical school will evaluate the qualifications of the respective departments if they apply for affiliation. This will embrace:

   a. A careful review of outlines of courses which are submitted by the chief of staff of the hospital under consideration.

b. Inspection and approval of the library facilities of proposed hospitals.

c. An evaluation of the departmental research program.

GRADUATE CREDIT

1. Staff members of affiliated hospitals who meet certain requirements will be accepted for graduate training. Exceptional students may register with graduate schools and pursue work leading to advanced degrees.

2. Credit for work done in residence or absentia rests with the chairman of the department in which the subject is regularly taught in the medical school. The possibility of an exchange of residents between the hospitals for a limited period is suggested.

3. Full schedules of study in affiliated hospitals are not recommended.

4. Requirements for advanced degrees for staff members of affiliated hospitals shall be those designated by the graduate schools of the various universities or medical schools.

A. C. FURSTENBERG, M.D., *Dean*
University of Michigan Medical School
Ann Arbor, Michigan

Memorial

Northwestern University Medical School will hold a memorial convocation for Dr. Sanford R. Gifford in Thorne Hall auditorium on the downtown Northwestern University Campus, Thursday, October 12, at 4:30 p.m. Dr. Irving S. Cutter will preside at the convocation and the Honorable Anan Raymond will speak.

OFFICIAL NOTICES

FORTY-NINTH ANNUAL CONVENTION

American Academy of Ophthalmology and Otolaryngology

OCTOBER 8 TO 12, 1944

Palmer House

Chicago, Illinois

Convention Call

Under the authority of the Constitution and with the approval of the Council, I herewith proclaim that the Forty-Ninth Annual Convention of the American Academy of Ophthalmology and Otolaryngology will convene at the Palmer House, Chicago, Illinois, on Sunday, October 8, 1944, and continue through Thursday, October 12, 1944.

LAWRENCE T. POST, M.D.
President

Attest:
WILLIAM L. BENEDICT, M.D.
Executive Secretary-Treasurer

Local Committees

RICHARD C. GAMBLE, M.D.
General Chairman

Reception Committee

Thomas D. Allen, M.D., *Chairman*
Howard C. Ballenger, M.D.
I. F. Barnett, M.D.
Beulah Cushman, M.D.
John F. Delph, M.D.
Earl B. Fowler, M.D.
Ira Frank, M.D.
Thomas C. Galloway, M.D.
Glenn J. Greenwood, M.D.
George P. Guibor, M.D.
L. G. Hoffman, M.D.
Paul H. Holinger, M.D.
Arlington Krause, M.D.
Vernon M. Leech, M.D.
John R. Lindsay, M.D.
William A. Mann, M.D.
G. Henry Mundt, M.D.
Frank J. Novak, Jr., M.D.
Samuel J. Pearlman, M.D.
Samuel Salinger, M.D.
George E. Shambaugh, Jr., M.D.

Committee on Arrangements

Arthur J. Coombs, M.D., *Chairman*
Carl H. Christoph, M.D.
Elmer Hagens, M.D.
A. R. Hollender, M.D.
George T. Jordan, M.D.
Alfred Lewy, M.D.
George S. Livingston, M.D.
Samuel J. Meyer, M.D.
Glen W. Nethercut, M.D.
Roy O. Riser, M.D.
Gail Soper, M.D.

Scientific Exhibit Committee

O. E. Van Alyea, M.D., *Chairman*
Georgiana Dvorak-Theobald, M.D., *Consultant*

Ladies' Committee

Mrs. Thomas D. Allen, *Chairman*
Mrs. Arthur J. Coombs, *Co-Chairman*

Council Meetings

The Council of the American Academy of Ophthalmology and Otolaryngology will convene at 9 o'clock, Sunday morning, October 8, in the Executive Office, Room No. 403, Palmer House, Chicago. There will also be meetings on Monday, Tuesday and Wednesday mornings of that week at 7:30 o'clock and on Thursday morning at 8 o'clock to transact such business as may come before it. Members desiring to be heard or to present business will confer with the Executive Secretary for appointments.

LAWRENCE T. POST, M.D.
President

Public Relations

Contributors to the scientific program are requested to send copies of their papers to Dr. Erling W. Hansen, Medical Arts Building, Minneapolis, Minnesota, Secretary for Public Relations, at the earliest possible moment.

LAWRENCE T. POST, M.D.
President

Exhibits

The Scientific and Technical Exhibits will open at 9 o'clock Sunday morning, October 8.

Registration

MISS CLARE MCGOVERN, *Registrar*

General registration will begin at 9 o'clock Sunday morning, October 8, in the Technical Exhibit Hall, Fourth Floor

1. Advance orders for conference tickets will be filled only for members whose 1944 dues have been paid.
2. Receipt for 1944 dues is essential for registration. Members without cards will be required to procure duplicates previous to registration.
3. Instructional conference tickets will be sold only to those registered and with badge.
4. A special information desk for foreign guests will be in charge of Miss Givens.
5. To facilitate registration and the transaction of such other business as is necessary at the Registration Desk, sections will be provided for:
   - A. Advance Orders.
   - B. Duplicate Receipt Cards.
     Payment of Dues.
     Filing of Academy Applications.
     Registration of Residents exempt from fee (previously arranged for by Head of Department of Institution where training).
     Registration of Academy candidates listed in July-August issue of the TRANSACTIONS and eligible for election at this meeting (exempt from fee).
   - C. Non-Members (fee $5.00).
     Candidates whose applications were received too late for listing in the July-August issue of the TRANSACTIONS (fee $5.00).
   - D. Purchase of Instructional Conference Tickets ($1.50 each period).
   - E. Purchase of tickets for Alumni Dinners and Annual Academy Dinner (price $3.50). Sale of tickets closes at 2 o'clock in the afternoon of the day of the dinner.
   - F. Purchase of instructional supplies (abstracts, covers, indexes, monographs).
     Orders for Bound Volume of TRANSACTIONS.
     Subscription to bi-monthly issues of TRANSACTIONS.
     Orders for Pathological Atlases—Ophthalmic and Otolaryngic.

Regulation Badge

The regulation badge secured at the time of registration is essential for admittance to all activities.

Please Note. — A badge will be furnished at the time of registration upon presentation of receipt card for 1944 dues. Members who fail to bring 1944 receipt card to Chicago will be required to obtain a duplicate previous to registration.

Ladies' Registration

The wives of members and lady guests are urged to register.

Ladies' Headquarters
Rooms 15 and 16, Club Floor

Guests

Physicians are welcome to attend the meeting of the Academy and to participate in all the activities. All guests, however, will be required to register and a fee of $5.00 will be charged. Academy candidates whose applications are filed previous to August 1 and whose names are published in the July-August issue of the TRANSACTIONS will be exempt from paying this $5.00 registration fee.

Residents Registering as Guests

Men in training in accepted residencies, or those now completing a full academic year (nine months) in basic study, registering as guests during the Academy convention period will be relieved of paying a guest fee only when the Department Head concerned officially requests this favor at least 30 days prior to the opening date of the convention.

New Business

Proposals for new activities often are presented too late during the Academy session to afford time for deliberation. Therefore, new items requiring Council action should be presented in writing to the Executive Secretary early so that the Committee concerned may have an opportunity to study the matter prior to presentation to the Council.

Advance Sale of Instructional Tickets

Members are urged to contract for instructional tickets by advance mail order. Please note instructions for reserving tickets on page 465 and on colored insert. Orders for tickets will be filled for members only whose 1944 dues have been paid.

Dues

A Reminder

Are your 1944 dues paid? Because of the second-class mail regulations, the TRANSACTIONS can be sent only to members in good standing.

Those who have not paid dues for 1944 are requested to remit to Dr. W. L. Benedict, Secretary-Treasurer, One Hundred First Avenue Building, Rochester, Minnesota.

Constitutional Changes in Fellowship Status

Life Fellows — 1945

Andrews, M. P. ..... Manitowoc, Wisconsin
Arbuckle, Millard F. (Col.) ..... U. S. Army
Ashley, T. W. ..... Kenosha, Wisconsin
Balentine, P. L. ..... Philadelphia, Pa.
Ballenger, H. C. ..... Winnetka, Illinois
Brooks, E. H. ..... Appleton, Wisconsin
Chapman, Vernon A. ..... Milwaukee, Wis.
Childs, H. A. ..... Creston, Iowa
Clark, L. H. ..... Rochester, N. Y.
Cott, C. C. ..... Buffalo, N. Y.
Davis, George E. ..... New York, N. Y.
Fulkerson, C. B. ..... Kalamazoo, Mich.
Fuller, T. E. ..... Texarkana, Texas
Gatewood, W. L. ..... New York, N. Y.
Hohf, Julius A. ..... Yankton, S. D.
Hompes, J. J. ..... Lincoln, Nebraska
Ingram, L. C. ..... Orlando, Florida
Kiehle, Frederick A. ..... Portland, Oregon
Lassen, Fritz ..... Pueblo, Colorado
Lewis, James P. (Comdr.) ..... U. S. Navy
Lloyd, A. W. ..... Hammond, Indiana
May, J. V. ..... Marinette, Wisconsin
Moulton, Everett C. ..... Fort Smith, Ark.
Nadeau, E. G. ..... Green Bay, Wisconsin
O'Brien, Steve A. ..... Mason City, Iowa
Pyle, Wallace ..... Jersey City, N. J.
Rector, A. E. ..... Appleton, Wisconsin
Rosenbaum, J. ..... Montreal, Canada
Rott, O. M. ..... Spokane, Washington
Rubendall, C. ..... Omaha, Nebraska
Scales, J. L. ..... Shreveport, Louisiana
Schlindwein, G. William ..... Erie, Pa.
Shields, J. M. ..... Denver, Colorado
Stevenson, Walter ..... Quincy, Illinois
Swetman, C. R. K. ..... Prescott, Arizona
Watson, H. deWitt ..... Binghamton, N. Y.
Woods, R. H. ..... La Salle, Illinois

Senior Fellows — 1945

Adams, D. S. .....Indianapolis, Indiana
Adams, Eldridge S. (Col.) .....U. S. Army
Adams, Frederick W. .....Seattle, Wash.
Allen, Charles E. ....Kansas City, Missouri
Altringer, A. N. .....Kansas City, Missouri
Auerbach, Julius .....New York, N. Y.
Baird, Charles G. (Col.).....U. S. Army
Bane, W. M. .....Denver, Colorado
Barlow, Roy A.....Winchester, Virginia
Bone, Herman D.....Gardner, Mass.
Bouvy, Harry M. .....Portland, Oregon
Braunlin, William H. .....Marion, Indiana
Brehmer, Harrison L....Albuquerque, N. M.
Brewer, John D. .....Dyersburg, Tenn.
Brown, John C. .....St. Paul, Minnesota
Butler, Robert H. .....Bellefontaine, Ohio
Camp, W. E.....Minneapolis, Minnesota
Chadwick, Ira B. .....Coffeyville, Kansas
Cheney, James W. .....Wichita, Kansas
Clark, I. G. .....Columbus, Ohio
Connor, C. E.....St. Paul, Minnesota
Craft, Kenneth L.....Indianapolis, Indiana
Davis, Ralph F. .....Portland, Oregon
De Boe, M. P.....Miami, Florida
Deichler, L. Waller.....Philadelphia, Pa.
De Weese, Clarence....Lexington, Kentucky
Dunlap, Lawrence G....Anaconda, Montana
Dyer, C. P.....St. Louis, Missouri
Ersner, Matthew S.....Philadelphia, Pa.
Fogarty, Charles.....St. Paul, Minnesota
Fox, C. H.....Kansas City, Missouri
Freedman, L. M.....Boston, Massachusetts
Gillett, Wilbur G.....Wichita, Kansas
Gillum, J. R.....Terre Haute, Indiana
Gittins, T. R.....Sioux City, Iowa
Gosney, Charles W.....Los Angeles, Calif.
Granger, W. B.....Emporia, Kansas
Greever, B. L.....Hutchinson, Kansas
Gregg, John B.....Sioux Falls, S. D.
Gross, Julius E.....Pittsburgh, Pa.
Gsell, J. F.....Wichita, Kansas
Haney, William P.....Omaha, Nebraska
Hansen, Erling W. .....Minneapolis, Minn.
Hawman, Erle G.....Philadelphia, Pa.
Hempstead, Bert E.....Rochester, Minn.
Hennessey, William W.....Salem, Mass.
Hoffman, Walter F.....Seattle, Washington
Hourn, George E.....St. Louis, Missouri
Jackson, J. D. .....Erie, Pa.
Judge, Harry V.....Albany, N. Y.
Keller, S. A.....Sioux Falls, S. D.
King, Charles R.....Toledo, Ohio
Klingner, Thomas O...Springfield, Missouri
Lemoine, A. N.....Kansas City, Missouri
Lewis, George A.....Roundup, Montana
Lillie, Harold I.....Rochester, Minnesota
Lukens, Robert McD.....Philadelphia, Pa.
McAlester, Andrew W. Jr..Kansas City, Mo.
McBean, George M.....Chicago, Illinois
McCullock, Robert J. P.....Toronto, Canada
Maggard, D. I.....Wichita, Kansas
Mercer, Ray.....Crossville, Tenn.
Middleton, Harry E.....Alton, Illinois
Miller, Hugh .....Kansas City, Missouri
Minton, William H.....St. Joseph, Missouri
Morsman, L. William...Hibbing, Minnesota
Myers, Elmer A.....Superior, Wisconsin
Myers, John L.....Kansas City, Missouri
Phelps, Kenneth A.....Minneapolis, Minn.
Post, Lawrence T.....St. Louis, Missouri
Prout, A. W.....Columbus, Ohio
Reim, Hugo.....St. Louis, Missouri
Repass, R. E.....Miami Beach, Florida
Rommel, John C.....Philadelphia, Pa.
Rooker, A. M.....Niagara Falls, N. Y.
Roth, Albert W.....Tulsa, Oklahoma
Rothschild, Harold J....St. Paul, Minnesota
Sacco, Anthony G.....Union City, N. J.
Salinger, Samuel.....Chicago, Illinois
Schenck, C. P.....Fort Worth, Texas
Sellers, L. M.....Dallas, Texas
Shiras, Howard H.....Cleveland, Ohio
Shoemaker, Ferdinand....Kansas City, Mo.
Smith, Arthur E.....Minneapolis, Minn.
Sneed, Carl M.....Columbia, Missouri
Spake, LaVerne B.....Kansas City, Kan.
Stewart, Walter E.....Terre Haute, Indiana
Taquino, George J.....New Orleans, La.
Trible, G. B. .....Washington, D. C.
Trimble, Clarence S.....Emporia, Kansas
Tripp, Leroy R.....Sioux City, Iowa
Wagers, Arthur J.....Philadelphia, Pa.
Weih, E. P.....Clinton, Iowa
Yazel, Herman E.....Kansas City, Mo.
Zinkhan, A. M.....Washington, D. C.

CONVENTION ACTIVITIES

President's Reception

RED LACQUER ROOM
Sunday, 5:00 p.m.

President Lawrence T. Post and Mrs. Post invite all members and their wives to a reception honoring Dr. and Mrs. Nelson Miles Black and the officers of the Academy.

Committee Programs

I. Conservation of Hearing
GRAND BALL ROOM
Sunday, 2:00 p.m.

II. Otosclerosis Study Group
PRIVATE DINING ROOM NO. 18
Sunday, 8:00 p.m.

III. Industrial Ophthalmology
GRAND BALL ROOM
Sunday, 8:00 p.m.

IV. American Orthoptic Council and American Association of Orthoptic Technicians
Joint Symposium
PRIVATE DINING ROOM NO. 14
Sunday, 8:00 p.m.

Honor Section Luncheon

PRIVATE DINING ROOM NO. 9
Monday, 12:45 p.m

(Honor Section Headquarters will be in the suite of the President, Dr. Thomas E. Carmody.)

Alumni Dinners

Monday, 6:30 p.m.

Smoker

Grand Ball Room, Monday, 9:00 p.m.

Faculty Luncheon

Private Dining Room No. 14
Tuesday, 12:45 p.m.

The President, Dr. Lawrence T. Post, and the other officers of the Academy invite all those presenting scientific papers, leaders of instructional conferences, participants in the motion picture sections and scientific exhibitors to be their guests at luncheon.

Annual Academy Dinner

Grand Ball Room, Tuesday, 7:00 p.m.
Informal

Annual Business Meeting

Grand Ball Room, Wednesday, 8:00 p.m.

Induction of New Members
Jackson Memorial Lecture

Grand Ball Room, Wednesday, 9:00 p.m.

EXAMINATION BY THE AMERICAN BOARD OF OTOLARYNGOLOGY

The next examination of the American Board of Otolaryngology will be held in Chicago at the Palmer House on October 4-5-6-7, 1944. For information, write Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.

EXAMINATION BY THE AMERICAN BOARD OF OPHTHALMOLOGY

Part I of the examination (for especially assigned candidates) will be held at the Edgewater Beach Hotel, Chicago, October 5 and 6. This examination will include the following subjects: Anatomy and Embryology, Perimetry, Therapeutics and Operations, Optics and Visual Physiology, Relation of the Eye to General Diseases, Pathology, Bacteriology and Immunology.

The practical and clinical examination, Part II, will be held at the Illinois Eye and Ear Infirmary October 7. This will include the following subjects: External Diseases—Slit Lamp; Ophthalmoscopy; Histology—Histopathology; Ocular Motility; Refraction—Retinoscopy; Practical Surgery.

S. JUDD BEACH, M.D., *Secretary*

Examination by the American Orthoptic Council

Examination for candidates for Council certificates will be held Saturday, October 7, at 2:00 p.m., at the Illinois Eye and Ear Infirmary.

LE GRAND HARDY, M.D.
President

American Association of Eye, Ear, Nose and Throat Society Secretaries

Private Dining Room No. 18
Wednesday, 5:00 p.m.

W. H. MORRISON, M.D., *President*

NOTICE TO MEMBERS:

The sixth annual meeting of the American Association of Eye, Ear, Nose and Throat Society Secretaries will be held on Wednesday evening October 11, 1944, at the Palmer House, Chicago, during the session of the American Academy. A dinner will be provided through the generosity of the Academy and ample time will be afforded for full discussion of the program.

The chief topic of the program will be "Extension Study Courses for Eye, Ear, Nose and Throat Societies," presented by Doctors William L. Benedict, Harry S. Gradle, Dean M. Lierle and A. D. Ruedemann. This subject is the brain-child of our President-Elect, Dr. Paul C. Craig of Reading, Pennsylvania, and should be of intense interest to all E.E.N.T. Societies and their secretaries. Full discussion from the floor is invited.

In addition, the directors of the various districts of the Association will report upon the results of the Monahan-Jones resolution passed at the 1943 meeting regarding the Murray-Wagner Act. A copy of this resolution was placed in the hands of the secretary of every E.E.N.T. Society in the United States and Canada. If you did not receive your copy please write the secretary.

Another item of interest upon the program will be the report of the committee appointed to draft a constitution and by-laws for the Association. This job has been finished at last and the document will be presented for your approval.

Also each secretary is invited to present his own suggestions and criticisms for the benefit of the Association. And if you need help in any problems facing you, as the secretary of an E.E.N.T. society, bring your questions to this meeting.

Plan now to attend this meeting for secretaries. We believe you will find it interesting and helpful. All secretaries are welcome.

KENNETH L. CRAFT, M.D., *Secretary*
1002 Hume-Mansur Building
Indianapolis 4, Indiana

Alumni Dinners

MONDAY, OCTOBER 9, 6:30 P.M.

$3.50

The facilities of the Academy management are placed at the disposal of alumni and other special groups. Tickets may be secured at the General Registration Desk before 2 p.m. Monday.

It may be necessary to make some changes in room schedules. Therefore, check bulletin board Monday afternoon.

NOTE: Please use private elevators, Northeast corner lobby floor to Club Building.

Dr. John F. Barnhill Alumni
Private Dining Room No. 4
C. H. McCaskey, M.D., Secretary
608 Guaranty Bldg., Indianapolis, Ind.

Dr. Joseph C. Beck Alumni
Private Dining Room No. 7
J. M. Sutherland, M.D., Secretary
662 Fisher Bldg., Detroit 2, Mich.

Bellevue Hospital Alumni
Private Dining Room No. 5
W. W. Wilkerson, Jr., M.D., Secretary
1022 Bennie-Dillon Building
Nashville, Tenn.

Brooklyn Eye and Ear Clinic
Private Dining Room No. 9
Capt. L. J. Croll, M.C. 0500837, Sec'y
Station Hospital, Fort Knox, Ky.

Dr. Wells P. Eagleton Alumni
Private Dining Room No. 3
William H. Hahn, M.D., Secretary
15 Lombardy St., Newark 2, N.J.

Harper Hospital Alumni
Room No. 795
William S. Gonne, M.D., Secretary
619 David Whitney Bldg., Detroit, Mich.

Illinois Eye and Ear Infirmary
Private Dining Room No. 17
H. I. McGarry, M.D., Secretary
904 W. Adams St., Chicago 7, Ill.

Institute of Ophthalmology — Presbyterian Hospital
Private Dining Room No. 11
Charles A. Perera, M.D., Secretary
70 E. 66th St., New York, N. Y.

Iowa Alumni
Private Dining Room No. 16
Dean M. Lierle, M.D., Secretary
University Hospital, Iowa City, Ia.

Mayo Clinic Alumni
Private Dining Room No. 1
Avery D. Prangen, M.D., Secretary
Mayo Clinic, Rochester, Minn.

Michigan Joint Alumni Group
Illinois Room
Canfield Society
James H. Maxwell, M.D., Secretary
800 First Nat. Bldg., Ann Arbor, Mich.
Parker Club
Ralph O. Rychener, M.D., Secretary
1750 Exchange Bldg., Memphis, Tenn.

Minnesota Alumni
Room No. 788
Frank Knapp, M.D., Secretary
Medical Arts Bldg., Duluth, Minn.

Dr. Harris P. Mosher Alumni
Private Dining Room No. 14
August L. Beck, M.D., Secretary
421 Huguenot, New Rochelle, N. Y.

New York Postgraduate Medical School and Hospital
Oval Room—Club Building
James W. Smith, M.D.
1016 Fifth Ave., New York 28, N. Y.

Dr. C. S. O'Brien Alumni
Private Dining Room No. 2
W. H. Maloy, M.D., Secretary
608½ W. Sheridan Ave., Shenandoah, Ia.

Pennsylvania Alumni—Otolaryngology
Club Building Lounge
Oram R. Kline, M.D., Secretary
514 Cooper St., Camden, N. J.

George Portman-Bordeaux Society
Private Dining Room No. 6
J. A. Flynn, M.D., President
1511 Rhode Island Ave. N.W.
Washington 5, D.C.

Washington University Alumni, St. Louis, Missouri
Crystal Room
Bennett Y. Alvis, M.D., Secretary
900 Carleton Bldg., St. Louis, Mo.

Wilmer Institute, the Johns Hopkins Hospital
Private Dining Room No. 8
Jack S. Guyton, M.D., Secretary
Wilmer Institute, Baltimore 5, Md.

Women Members
Private Dining Room No. 15
C. Souter Smith, M.D., Chairman
513 Holland Bldg., Springfield, Mo.

PROGRAM IN BRIEF

1944 Meeting — Chicago

SUNDAY, OCTOBER 8

9:00 a.m.—Council Meeting—Room 403
9:00 a.m.—Registration—Exhibit Hall, Fourth Floor
9:00 a.m.—Official Opening of Exhibits—Scientific and Technical
10:00 a.m.—Ladies' Registration—Rooms 15 and 16—Club Floor
10:00 a.m.—Meeting of American Committee on Optics and Visual Physiology—Private Dining Room No. 1
10:00 a.m.—Meeting of Entire General Committee on the Conservation of Hearing—Private Dining Room No. 18
10:00 a.m.—Meeting of Executive Committee on Industrial Ophthalmology—Private Dining Room No. 2
10:00 a.m.—Meeting of Council Committees
10:00 a.m.—Meeting of Honor Section—Private Dining Room No. 9
2:00 p.m.—Scientific Program — Conservation of Hearing — Grand Ball Room
5:00 p.m.—President's Reception—Red Lacquer Room
8:00 p.m.—Scientific Program — Otosclerosis Study Group — Private Dining Room No. 18

8:00 p.m.—Scientific Program — Industrial Ophthalmology—Grand Ball Room
8:00 p.m.—Joint Symposium — American Orthoptic Council and American Association of Orthoptic Technicians Private Dining Room No. 14

MONDAY, October 9
7:30 a.m.—Council Breakfast Meeting —Room 403
8:30 a.m.—Instructional Conferences—Seventh Floor
10:00 a.m.—Instructional Conferences—Seventh Floor
11:30 a.m.—Instructional Conferences—Seventh Floor
12:45 p.m.—Honor Section Luncheon — Private Dining Room No. 9
2:00 p.m.—Scientific Sections — Joint Session—Grand Ball Room
6:30 p.m.—Alumni Dinners
9:00 p.m.—Smoker—Grand Ball Room

TUESDAY, OCTOBER 10
7:30 a.m.—Council Breakfast Meeting —Room 403
8:30 a.m.—Instructional Conferences—Seventh Floor
10:00 a.m.—Instructional Conferences—Seventh Floor
11:30 a.m.—Instructional Conferences—Seventh Floor
12:45 p.m.—Faculty Luncheon—Private Dining Room No. 14
2:30 p.m.—Section on Otolaryngology—Scientific Papers—Red Lacquer Room
2:30 p.m.—Section on Ophthalmology — Motion Pictures — Grand Ball Room
7:00 p.m.—Annual Academy Dinner — Grand Ball Room

WEDNESDAY, OCTOBER 11
7:30 a.m.—Council Breakfast Meeting —Room 403
8:30 a.m.—Instructional Conferences—Seventh Floor
10:00 a.m.—Instructional Conferences—Seventh Floor
11:30 a.m.—Instructional Conferences—Seventh Floor
2:00 p.m.—Section on Ophthalmology —Scientific Papers — Red Lacquer Room
2:00 p.m.—Section on Otolaryngology —Motion Pictures — Grand Ball Room
5:00 p.m.—Dinner Meeting of American Association of Eye, Ear, Nose and Throat Society Secretaries—Private Dining Room No. 18
8:00 p.m.—Business Meeting — Grand Ball Room

THURSDAY, OCTOBER 12
8:00 a.m.—Council Breakfast Meeting —Room 403
9:00 a.m.—Section on Ophthalmology—Scientific Papers—Red Lacquer Room
9:00 a.m.—Section on Otolaryngology — Scientific Papers — Grand Ball Room
12:45 p.m.—Luncheon for Technical Exhibitors—Crystal Room
2:00 p.m.—Scientific Sections — Joint Session — Motion Pictures —Presentation of New Instruments — Demonstration of New Methods—Grand Ball Room

SCIENTIFIC PROGRAMS

Conservation of Hearing

GRAND BALL ROOM
Sunday, 2:00 p.m.

DEAN M. LIERLE, M.D., *Chairman*

1. Physiologic Effect of Pressure Changes with Reference to Otolaryngology
   A. R. Behnke, Comdr., M.C., U.S.N. Bethseda, Md.
   BY INVITATION
2. Status of the Problem of Aviation Deafness
   Paul A. Campbell, Lt. Col., M.C. A.U.S., Randolph Field, Texas
3. The Effect of Barometric Pressure Change Upon Hearing
   Clair M. Kos, Maj., M.C., A.U.S. Randolph Field, Texas
4. Effects of Gun Blast Upon Hearing
   Willard Machle, Col., M.C., A.U.S. Fort Knox, Ky.
   BY INVITATION

5. Aero-Otitis Media and Auditory Acuity Loss in Submarine Escape Training
   C. W. Shilling, Capt., M.C., U.S.N. New London, Conn.
   BY INVITATION
6. Effect of Aircraft Noise Upon Hearing
   Ben Senturia, Capt., M.C., A.U.S. Randolph Field, Texas
7. The Vocational Adjustment of the Aurally Handicapped
   M. M. Walter
   Director of Pennsylvania State Rehabilitations Bureau
   Harrisburg, Pa.
   BY INVITATION

Industrial Ophthalmology

GRAND BALL ROOM
Sunday, 8:00 p.m.

ALBERT C. SNELL, M.D.
Chairman

1. Eye Protective Equipment (illustrated).
   Glen H. Harrison, M.D., member of the Joint Committee on Industrial Ophthalmology.
2. The Connecticut Experience with Eye Problems in Small Industries.
   Joseph Lo-Presti, M.D., Assistant Surgeon (R), U. S. Public Health Service, on assignment to National Society for the Prevention of Blindness.
   BY INVITATION
3. Illumination and Industrial Visual Tasks (illustrated).
   Allen K. Gaetjens, Research Engineer, Nela Park, General Electric Co., Cleveland, Ohio.
   BY INVITATION
4. Placement of Blind in Industry (illustrated).
   Hazel Hurst, President, Hazel Hurst Foundation, Los Angeles, Calif. Placement Officer for Blind at Lockheed Aircraft, Los Angeles, Calif. (To be introduced by Meyer Wiener, M.D., Coronado, Calif., Ophthalmic Consultant to Vice Admiral McIntyre, Surgeon General, United States Navy.)

American Orthoptic Council
and
American Association of Orthoptic Technicians
Joint Symposium

PRIVATE DINING ROOM NO. 14
Sunday, 8:00 p.m.

LEGRAND HARDY, M.D.
Chairman

1. Follow-Up in Orthoptics.
   Miss Jean Robinson, Memphis, Tenn.
   Discussion opened by Edward C. Ellett, M.D., Memphis, Tenn.
2. Analysis of Failures in the Treatment of Squint.
   Frank Costenbader, M.D.
   Washington, D. C.
   Discussion opened by Miss Marjorie Enos, New York, N. Y.
3. Orthoptic Fictions and Misconceptions.
   Miss Louisa Wells, Washington, D. C.
   Discussion opened by Walter B. Lancaster, M.D., Boston, Mass.

Otosclerosis Study Group

PRIVATE DINING ROOM NO. 18
Sunday, 8:00 p.m.

GEORGE E. SHAMBAUGH, JR., M.D.
Chairman

1. Annual Summary of Results of the Fenestration Operation as Contributed by Members of the Group.
2. Symposium on Experimental Fenestration of the Labyrinth
   John R. Lindsay, M.D.
   Henry B. Perlman, M.D.
   George E. Shambaugh, Jr., M.D.
   Chicago, Ill.
   Dorothy Wolff, Ph.D.
   Boston, Mass.
3. Further Evolution of the Fenestration Operation
   Julius Lempert, M.D.
   New York, N. Y.

SCIENTIFIC SECTIONS

Monday, October 9, 1944, 2 p.m.

Joint Session

GRAND BALL ROOM

Presiding Officer:

President, LAWRENCE T. POST, M.D.

Secretaries in Charge

Ophthalmology:
Algernon B. Reese, M.D.

Otolaryngology:
O. E. Van Alyea, M.D.

Call to Order.....The President

Announcements. The General Chairman
Richard C. Gamble, M.D., Chicago, Ill.

Introduction of the Guest of Honor

Introduction of Special Guests

President's Address
Lawrence T. Post, M.D., St. Louis, Mo.

Address of the Guest of Honor
Nelson M. Black, M.D., Miami, Fla.

Empirical Treatment of Nyctolopia by a General Practitioner in Central America in 1912—A Reminiscence

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Symposium: *Head and Face Pain*

OPHTHALMOLOGY
Walter I. Lillie, M.D., Philadelphia, Pa.

OTOLARYNGOLOGY
Arthur W. Proetz, M.D., St. Louis, Mo.

MEDICAL
Bayard T. Horton, M.D., Rochester, Minn.
BY INVITATION

NEUROLOGY
Lewis J. Pollock, M.D., Chicago, Ill.
BY INVITATION

Authors of Papers

Please be prepared to submit papers to the recorder immediately following presentation.

There will be discussion of papers presented at the scientific sections Tuesday, Wednesday and Thursday.

Section on Otolaryngology

TUESDAY, OCTOBER 10, 1944

2:30 p.m.

RED LACQUER ROOM

Presiding Officer:

C. STEWART NASH, M.D.
First Vice President

Secretary:

O. E. VAN ALYEA, M.D.

An Analysis of Colds in Industry
JOSEPH H. KLER, M.D.
New Brunswick, N. J.

Discusser:
THEODORE E. WALSH, M.D.
St. Louis, Mo.

A critical analysis has been made of a "cold" survey among 5,500 employees of Johnson & Johnson in New Brunswick and Chicago, from July, 1942, to February, 1944. The survey endeavored to learn something of the incidence of colds among men and women employees as affected by age, sex, type of work, shift, and surrounding physical conditions of heating and ventilation. Such general outside factors as time of year, temperature, humidity and epidemics, were correlated both with incidence and severity of colds. In addition a careful study was made of all of the more severe colds. Individual case histories were analyzed as to past medical history, individual susceptibility, dietary factors, self medication, health habits, use of vitamins, menstrual history, etc.

Slides of composite graphs and charts are available.

Deafness, Its Causes and What Can Be Done About It
M. H. LURIE, M.D.
Boston, Mass.

Discusser:
W. J. McNALLY, M.D.
Montreal, Canada

Deafness, its diagnosis and treatment is receiving more attention these days due to intense war research being done.

As a result of the improved technic for the treatment of otosclerosis the otologist feels that he can do more for the deaf than formerly. Vitamins and various drugs are also being used for the purpose of increasing the hearing of the deaf. The causes of deafness, its prevention and its treatment will be discussed from the point of view of what can we do for the deaf person and what is the outlook for the future.

A New Sinus Syndrome
SAM E. ROBERTS, M.D.
Kansas City, Mo.

Discusser:
C. STEWART NASH, M.D.
Rochester, N. Y.

The syndrome to be discussed concerns nonsuppurative sinusitis. The following symptoms are usually present:

1. Nasal obstruction without visual pathology
2. Headache
3. Postnasal discharge without the usual signs of infection
4. Fatigue without organic findings elsewhere in the body

This is frequently the most difficult type of sinus trouble to treat because surgery is rarely indicated. The patient must be investigated from a nutritional and biochemical point of view.

This report is based on the experience over a period of seven years and more than 4,000 cases have been managed under this regime.

Methods of investigation will be given and the office treatment will be emphasized in detail.

Treatment of Laryngeal and Tracheal Stenosis
JOHN B. ERICH, M.D.
Rochester, Minn.

BY INVITATION

Discussers:
HENRY B. ORTON, M.D.
Newark, N. J.
GORDON B. NEW, M.D.
Rochester, Minn.

The treatment of cicatricial stenosis of the larynx and up through the trachea is considered in three stages:

1) Surgical eradication of the stenosis by excision of the intralaryngeal scar tissue through a thyrotomy incision, and lining the freshly denuded surface with a thin Thiersch skin graft. The latter procedure offers a rapid method of epithelialization for the laryngeal lumen and, in turn, tends to reduce the subsequent development of scar tissue.

2) Maintenance of the laryngeal opening during the period of contracture by means of an obturator prepared from one of the acrylic dental materials. In contrast to rubber obturators, those made of an acrylic material do not change shape or become elongated; they eliminate the formation of granulation tissue within the larynx or trachea, and obviate the necessity of frequent laryngeal examinations and the changing of obturators. Moreover, acrylic obturators are nonporus and, therefore, perfectly clean and odorless.

3) Removal of the obturator and closure of the tracheal opening.

Section on Ophthalmology

WEDNESDAY, OCT. 11, 1944

2 p.m.

RED LACQUER ROOM

Presiding Officer:
LAWRENCE T. POST, M.D.
President

Secretary:
ALGERNON B. REESE, M.D.

Choice of Operation in Eyes with Primary Glaucoma and Cataracts
JACK S. GUYTON, M.D.
Baltimore, Md.

Discussers:
PAUL A. CHANDLER, M.D.
Boston, Mass.
J. HEWITT JUDD, M.D.
Omaha, Neb.

An analysis of 44 cases of eyes with primary glaucoma complicated by cataracts shows the results obtained by various surgical procedures. This small series of cases indicates such differences in the reaction of certain types of primary glaucoma, complicated by cataracts, to different operative approaches that the following conclusions appear justified:

1. If the glaucoma is noncongestive and cannot be controlled with miotics, an anterior fistulizing operation should be performed prior to cataract extraction.

2. If the glaucoma is noncongestive but can be controlled with miotics, or if it is chronic congestive, a combined cataract extraction should be the initial procedure and is likely to be the only operation necessary.

An interesting side observation in this series was that making a cataract incision through an anterior fistulizing bleb did not necessitate a subsequent glaucoma operation more often than making the cataract incision in front of the bleb.

Configurational Aspects of Visual Acuity (Report of research on Visual Acuity sponsored by the Academy through the joint Committee on Optics and Visual Physiology)

CARL R. BROWN, Ph.D.
Ann Arbor, Mich.
BY INVITATION

Experimental evidence is presented which is interpreted as indicating the inadequacy of the traditional "atomistic" approach to the problem of evaluating visual acuity, and its associated working concept of the "two-point limen," or resolving power. Test patterns of several kinds have been used in "acuity" measurements of the same group of eyes and comparisons of results are given. A test pattern consisting of a single elliptical spot is found to serve as well, according to available criteria as a test pattern, such as the Landolt ring, based upon the two-point or resolving power concept. The elliptical spot is both simpler (physiologically and psychologically) and productive of less ambiguous results than test patterns commonly in use because: (a) it is geometrically simpler, and more nearly free of the "inference" error, and (b) its "phenomenal" shape is its critical aspect, whereas a resolution test depends upon two spots (practically so, at any rate, in the Landolt ring, Snellen E, etc.) the "phenomenal" shapes of which vary greatly but are not directly critical for the test.

Special attention is also given to the question of the number of judgments required of the subject to establish his acuity to any desired degree of accuracy, and to the conditions determining the degree of **uncertainty** of the "phenomenal" test pattern. There is reason to believe that the steepness of the curve of correct judgments plotted against the independent variable of the test(distance, size, shape, degree of blurring, illumination intensity, etc.) is a significant aspect of vision, or, in other words that visual acuity, in the broadest sense is not a one-dimensional magnitude.

Enucleation and Substitute Operations Discussion of Factors Important for Better Cosmetic Effect

1 — Personal Experiences

HARRY S. GRADLE, M.D.
Chicago, Ill.
CECIL S. O'BRIEN, M.D.
Iowa City, Iowa
DANIEL B. KIRBY, M.D.
New York, N. Y.

2 — The Effect of Enucleation on the Orbit

RAYMOND L. PFEIFFER, M.D.
New York, N. Y.

Removal of the eyeball during the growth period stops the development of the orbit and leads to a shrinking of its capacity as observed by roentgenography. If performed later in life, the reduction is less evident and requires a longer period to become manifest. Implantation of spheres tends to maintain the orbital pressure and counteracts the contracting process. Roentgenological evidence will be presented.

Migration of the implant, whether due to faulty technic used at operation or to an unavoidable circumstance, may be quite unfortunate and may be demonstrated by the roentgen ray.

3 — Plastic Ocular Prostheses in Unusual Sockets

BENJAMIN F. SOUDERS
Capt., M.C., A.U.S.
Philadelphia, Pa.
BY INVITATION
HARVEY J. FORESTNER
Capt., D.C., A.U.S.
St. Louis, Mo.
BY INVITATION

The application of plastic (acrylic resin) compounds in fabrication of ocular prostheses by an impression-molding technic is described and appraised. Emphasis is placed particularly upon the creation of prostheses for deformed and unusual sockets. Lantern slides will be shown of illustrative cases.

Surgical Management of Glaucoma in Correlation with Gonioscopy and Biomicroscopy

SAMUEL J. MEYER, M.D.
Chicago, Ill.

PAUL STERNBERG, M.D.
Chicago, Ill.

BY INVITATION

Discussers:

ROBERT MASTERS, M.D.
Indianapolis, Ind.

JOHN M. McLEAN, M.D.
New York, N. Y.

Gonioscopy and biomicroscopy can be of limited aid in the surgical management of the various types of glaucoma. These clinical instruments of investigation present evidence with which the ophthalmologist may better evaluate the type, site and means to the surgical approach of the individual glaucoma problem.

Section on Ophthalmology

THURSDAY, OCTOBER 12, 1944
9 a.m.

RED LACQUER ROOM

Presiding Officer:

LAWRENCE T. POST, M.D.
President

Secretary:

ALGERNON B. REESE, M.D.

Extraction of Senile Cataract
A Statistical Comparison of Various Technics and the Importance of Preoperative Survey

WILLIAM F. HUGHES, JR., M.D.
Baltimore, Md.
and
WILLIAM C. OWENS, M.D.
Baltimore, Md.

BY INVITATION

Discussers:

JOHN H. DUNNINGTON, M.D.
New York, N. Y.

FREDERICK A. DAVIS, M.D.
Madison, Wis.

During the past eighteen years at the Wilmer Institute, the early technic consisting of a combined extracapsular cataract extraction without sutures has undergone a series of gradual but radical changes; viz., the use of a conjunctival flap closed with sutures, intracapsular extraction, corneoscleral sutures, and finally a peripheral iridotomy with preservation of a round pupil. Statistical analysis of 2,100 extractions of uncomplicated senile cataract during this period shows a steady reduction in the incidence of postoperative complications and the recovery of better final vision coincident with each modification in the original technic.

An analysis of individual factors associated with the various technics reveals the following significant correlations: (a) The amount of capsule and lens cortex remaining after an extracapsular extraction is directly related to the development of postoperative uveitis and secondary glaucoma. (b) Loss of vitreous predisposes to postoperative detachment of the retina, persistent vitreous opacities, and secondary glaucoma. (c) Corneoscleral sutures reduce the incidence of incomplete closure of the wound, prolapse of iris, and the amount of final astigmatism. (d) Preservation of the round pupil reduces the likelihood of vitreous loss and postoperative anterior chamber hemorrhage.

The preoperative surveys of the general medical status of the patients in this series have been analyzed and correlated with the incidence of postoperative complications. After extracapsular extractions only, the patients with a known inadequately treated focus of infection (usually in the nose and throat or teeth) had a higher incidence of postoperative uveitis than those in which a known focus had been adequately treated. Syphilis, either treated or untreated, had no effect on postoperative complications. Large anterior chamber hemorrhages were more frequent in patients with diabetes of over eight years' duration or requiring over 30 units of insulin daily, but there is no correlation between the height of the blood sugar and postoperative complications. Systemic hypertension is not associated with any serious complication of cataract extraction.

The Basic Principles of Retinal Detachment Operations with Special Reference to the Eyeball Shortening Operation

DOHRMANN K. PISCHEL, M.D.
San Francisco, Calif.

Discussers:

CONRAD BERENS, M.D.
New York, N. Y.

FRANCIS H. ADLER, M.D.
Philadelphia, Pa.

In the fifteen years that have passed since ophthalmologists in large number have been operating and curing cases of retinal detachments, certain principles originally considered as theories have now come to be recognized as indisputable facts.

The first and most important of these is that to cure a retinal detachment, the hole or tear which is causing the retinal detachment must be closed or walled off by a solid line of water tight choroido-retinal adhesions from the remainder of the fundus. To produce this condition is the aim of all operations for curing retinal detachments.

To be successful, all types of retinal detachment operations must achieve the following four results:

1. An exudative choroiditis must be stirred up.
2. This must be done at the proper place.
3. The retina must be allowed to settle back to its normal position so that the hole or tear will touch this exudate and be sealed by it.
4. To do this, the subretinal fluid must be drained away, allowed to absorb, or both.

Any operation which will do this can cure retinal detachments.

The usual operations are well known and will not be discussed.

The plain so-called idiopathic retinal detachments are so regularly curable, that no further mention is necessary. Difficult cases warrant special discussion.

Aphacic eyes are notoriously difficult to cure, and for them there is perhaps need for a special operation, such as the Lindner's Undermining Operation.

Other difficult cases, such as unsuccessfully operated eyes, eyes with shrunken retinae, or with fine vitreous strands, can sometimes be helped, and cured by the eyeball shortening operation.

This operation is to be described and discussed in detail. Practical points in its execution, as well as accidents that may occur, will be brought out. Three successful cases will be described.

Operative Treatment of Heterophoria

C. DWIGHT TOWNES
LT. COL., M.C., A.U.S.
San Antonio, Texas

Discussers:

JAMES W. WHITE, M.D.
New York, N. Y.

BRITTAIN F. PAYNE, MAJ.
M.C., A.U.S., Randolph Field, Texas

The results of the operative treatment of 136 patients with heterophoria are given. This group included 66 patients with exophoria, 51 patients with esophoria and 19 patients with hyperphoria. All the patients were healthy young men between the ages of 18 and 26 years. An effort was made to reduce the phorias to the following requirements: for hyperphoria, less than two prism diopters, for esophoria, less than eight prism diopters, and for exophoria, less than six prism diopters. These figures represent measurements with the Maddox rod at twenty feet. The objective was obtained in approximately 80 per cent of the cases. This is a preliminary report based upon early results averaging only three months from the time of operation.

Iridocyclitis, Complicating Cataract, and Retinal Separation: Their Interrelationship

EDMUND B. SPAETH, M.D.
Philadelphia, Pa.

Discussers:

THEODORE L. TERRY, M.D.
Boston, Mass.

S. RODMAN IRVINE, MAJ. M.C.
A.U.S., Fresno, Calif.

The Durerian triad of war, famine, and pestilence has as a modern homonymy these three allied conditions, iridocyclitis, complicating cataract, and retinal separation. Each result in destruction. One seems to depend upon the other, not only in constant variations in sequence of development but also in the causation of one by the other, here also with variable sequence. These facts are well known, so well, in fact, that one is inclined to be contemptous of one of them, especially when it appears at the moment alone. Our results from the surgery of retinal separation are still far from certain, and the complications seen not uncommonly after extractio lentis for a complicating cataract are rather well known. A survey

of 25 consecutive cases of this complex from the file of private patients showed an uncomfortably high incidence of serious complications. Ten consecutive clinic cases, when reviewed, were in degrees of serious impairment of vision in an even worse terminal state. These statements and the two mentioned truisms seem adequate reasons for the presentation of several of these cases mentioned with a brief analysis of the pathological complex. The underlying bacteriologic or metabolic etiology is not to be considered simply the interdependence of one upon the other.

Section on Otolaryngology

THURSDAY, OCTOBER 12, 1944
9 a.m.

GRAND BALL ROOM

Presiding Officer:

C. STEWART NASH, M.D.
First Vice President

Secretary:
O. E. VAN ALYEA, M.D.

Penicillin in Acute Surgical Mastoidectomy Wounds with Primary Suture

LEIGHTON F. JOHNSON, M.D.
Boston, Mass.
and
LOUIS WEINSTEIN, Ph.D., M.D.
Boston, Mass.
BY INVITATION

Discusser:

HENRY L. WILLIAMS, M.D.
Rochester, Minn.

Twenty-three cases of acute suppurative mastoiditis complicating 22 cases of scarlet fever and 1 case of measles were treated by simple mastoidectomy and the local application of penicillin through a No. 5 ureteral catheter implanted into the antrum. The dosage of the antibiotic agent found to be effective after some trial was 120,000 units given in doses of 10,000 units every eight hours for four days. On this regimen 19 of the patients needed no further therapy and have remained well as late as two months after operation. In the 4 patients who developed aural discharges in from two to five days after the cessation of treatment a ureteral catheter was reinserted through a very small incision in the postaural wound into the mastoid antrum and 120,000 units of penicillin given again for 4 days; every one of these cases was completely cured by this second course of therapy. No case had to be operated upon again.

Treatment of Sinusitis by Penicillin

I. JEROME HAUSER
Maj., M.C., A.U.S.
Springfield, Mo.
and
WALTER P. WORK, Capt., M.C., A.U.S.
Springfield, Mo.

Discusser:

HENRY L. WILLIAMS, M.D.
Rochester, Minn.

Various types of sinus infections were treated with penicillin. Among the cases treated were acute suppurative pansinusitis with complications, acute exacerbations of chronic suppurative sinusitis with complications, uncomplicated chronic suppurative sinus disease, and allergic rhinosinusitis with superimposed infection. In several cases the sinusitis was complicated by an epidural or brain abscess. Because of the scarcity of penicillin, uncomplicated acute suppurative sinusitis was not treated by this drug.

Some of the cases were treated with penicillin alone, or by penicillin and sulfonamides, while others were operated on first and then penicillin was administered. The penicillin was usually administered intramuscularly but in a few cases of subacute maxillary sinus infection the drug was instilled directly into the antrum. Cultures of the organism were obtained when possible, and the sensitivity of the organism to penicillin was determined. The course of the sinus infection was followed by frequent roentgenographic examinations, cultures and careful clinical observation. The results obtained in the treatment of the various cases of sinusitis with penicillin will be evaluated, and the indications for its use will be discussed.

Speech and Voice Defects Due to Oral and Laryngeal Anomalies

JAMES S. GREENE, M.D.
New York, N. Y.

Discussers:

MAXWELL D. RYAN, M.D.
New York, N. Y.

CLARENCE T. SIMON
Evanston, Ill.

BY INVITATION

DORIS YOAKUM TWITCHELL, Ph.D.
DeKalb, Ill.

BY INVITATION

Oral anomalies and laryngeal involvements often have an effect on speech and voice disproportionate to the severity of the actual structural abnormality. A minor abnormality, such as a foreshortened palate, may produce a major voice defect and in turn, a serious personality disorder. On the other hand, complete extirpation of the tongue may result in a comparatively slight speech defect. Slides and recordings illustrate various tongue, palate and laryngeal conditions and associated speech and voice involvements. Surgical procedures and speech therapy are discussed.

Late and Definite Treatment of War Injuries of the Face and Neck

MICHAEL LEWIN, Capt., M.C., A.U.S.
Brigham City, Utah

Discussers:

V. H. KAZANJIAN, M.D.
Boston, Mass.

JOHN B. ERICH, M.D.
Rochester, Minn.

BY INVITATION

This paper deals with various problems in reconstructive surgery of gunshot wounds of the face and neck. Various types of commonly encountered facial injuries are discussed with presentation of slides showing cases before, during and after treatment. Plastic procedures are described including the skin grafting for soft tissue losses and mucous membrane replacement by epithelial inlays. The treatment of gunshot wounds of the facial skeleton and their sequelae (like nonunion and oroantral fistula) is outlined.

MOTION PICTURES

PLEASE NOTE

(A) New instruments will be shown at the Motion Picture Sessions. Appointments for showing new instruments must be made with the Secretary. In order that proper publicity may be given, member showing a new instrument should prepare a brief description of that instrument and hand this to the Secretary in charge, who will see that it is turned over to the Editorial Board for publication in the TRANSACTIONS.

(B) Exhibitors showing films must acquaint the operator with instructions prior to the time of exhibiting.

(C) Due to legal restrictions on account of fire hazard, only safety film can be shown at the Palmer House at the October convention of the Academy. This applies to 16 mm. and to 35 mm. film, and particularly to sound film. Please be sure that the film you use is safety film.

Motion Pictures, Ophthalmology

TUESDAY, OCTOBER 10, 1944

2:30 p.m.

GRAND BALL ROOM

Presiding Officer:

HAROLD F. WHALMAN, M.D.
Second Vice President

Secretary:
ALGERNON B. REESE, M.D.

FILM NO. 1

The Scleral Flap Cataract Operation

CHARLES HYMES, M.D.
Minneapolis, Minn.

This film illustrates the intracapsular cataract extraction with peripheral iridectomy through the scleral flap incision as devised by the author.

Advantages of this method are:

1. Scleral suture may be placed prior to opening the eyeball.
2. Suture may be firmly tied without buckling the cornea.

3. An interval between suture of 2 mm. or more allows adequate space for completion of incision without fear of cutting the suture.
4. The scleral flap provides an excellent point of fixation.

Technic:

A 3 mm. conjunctival flap with Tenon's capsule is dissected to the limbus over the upper half of the cornea. A wedge shaped scleral flap with its base at the limbus is made with a Lonsgaard knife. The incision starts 1½ mm. above the upper limbus, the knife being directed toward the anterior chamber and stops just short of opening the eyeball. A silk suture is then placed through the conjunctiva and tip of scleral flap, and to the opposing point of sclera and conjunctiva above. The loop is drawn aside and the incision completed with keratome and scissors or Graefe knife.

The lens is grasped below the equator with an Arruga capsule forceps and pressure applied at the lower limbus with a muscle hook. As the lens begins to present through the pupil, pressure from below is correspondingly shallowed. No traction is made until the lens is well through the pupil, when side to side as well as anterior traction is exerted with the capsule forceps. Upon delivery of the lens the scleral suture is firmly tied and two or more auxiliary conjunctival sutures are placed.

FILM NO. 2

Errors of Refraction

WILLIAM A. MANN, M.D.
Chicago, Ill.
and
E. GEORGE NADEAU, M.D.
Green Bay, Wis.

A kodachrome moving picture film produced with the cooperation of the Department of Visual Education of Northwestern University Medical School and intended to clarify certain principles of physiological optics to undergraduate medical students, students of physics, and interested lay groups. Demonstrations are entirely by means of animated drawings.

The first section deals with the optical properties of lenses. Simple refraction, the effect of prisms on light rays, and the construction of an effect produced by convex and concave spheres and cylinders are illustrated.

The second section deals with the human eye. The various anatomical structures of the eye and orbit are pointed out. The formation of a retinal image in an emmetropic eye and the act of accommodation are demonstrated. Presbyopia, myopia, hyperopia, and astigmatism and the method of their correction with lenses are shown.

FILM NO. 3

Severe Symblepharon

WENDELL L. HUGHES, M.D.
Hempstead, N. Y.

Illustration of the technic of several procedures is shown in this film.

The original injury was caused by a splash of molten metal striking the left eye. The patient was first seen when the acute inflammation had subsided. The nasal half of the lower lid was destroyed and its skin had grown up over the cornea completely covering it. The fornix in this area and the opposing part of the upper fornix was obliterated.

The first step was to reconstruct the fornices above and below by the use of buccal mucous membrane flap.

The nasal end of the lower lid was rebuilt utilizing the opposing portion of the upper tarsoconjunctiva. A lash transplant was done later to provide lashes for the nasal end of the lower lid.

An unsuccessful attempt was made to save the eye. An enucleation with gold ball implant was done. The socket was small so a skin graft was utilized to reconstruct the socket so the patient could wear an artificial eye.

The original condition, the technic of the various steps and the final condition are shown.

FILM NO. 4

Congenital Nevus Pigmentosa of the Lids
Removal and Repair

WENDELL L. HUGHES, M.D.
Hempstead, N. Y.

This film depicts the removal of most of one upper and lower lid because of involvement with congenital nevus pigmentosa. The method of reconstruction of the internal layer (conjunctiva and tarsus) and of the external layer skin and lashes by utilizing the uninvolved portions of the conjunctiva and upper tarsus and the remaining lid skin to form a complete blepharorrhaphy is shown.

The transplantation of a hair-bearing strip of skin from the brows was done while the blepharorrhaphy was still intact. The incision for the new lid fissure was made between these two rows of lashes.

The original condition, the various stages of the procedure and the final condition are shown.

FILM NO. 5
Cataract Extraction
JACK S. GUYTON, M.D.
Baltimore, Md.

This film illustrates the technic of extracting senile cataracts most often employed in the Wilmer Ophthalmological Institute of the Johns Hopkins Hospital. During preparation of the eye for operation the pupil is dilated with 2 per cent homatropine and 1 per cent neo-synephrine. Akinesia is attained by the O'Brien or the van Lint method, and 1.5 cc. of 2 per cent novocaine with adrenalin 1:20,000 is injected retrobulbarly. A Parke lid speculum is inserted. The assistant grasps the lower bulbar conjunctiva and rotates the eyeball downward. A superior rectus bridle suture is inserted. At this stage, either two preplaced corneoscleral sutures of the type described by McLean are inserted and followed by a Graefe section, or a Graefe section is performed just back of the limbus and followed by the placement of two corneoscleral sutures. In either event the assistant removes the fixation forceps immediately after the section is completed. A peripheral iridotomy is performed at 12 o'clock. The lens is grasped with Arruga or Castroviejo forceps just anterior to the lower equator and delivered in the capsule by tumbling, utilizing a combination of traction and slight rotation with the forceps and counterpressure with a Budapest instrument. As an alternative, a Castroviejo suction tip connected to an electric suction pump is often used instead of forceps for delivering fully mature or hypermature cataracts. After the lens is delivered the iris is reposited and the corneoscleral sutures are tied. A solution of ¼ per cent eserine is instilled twice. If preplaced McLean corneoscleral sutures have been used, three conjunctival sutures are inserted and tied to hold the small conjunctival flap in place. The superior rectus suture and lid speculum are removed and a binocular dressing is applied.

FILM NO. 6
Extraocular Muscles
ARNO E. TOWN
Lt. Comdr., M.C., U.S.N.R.
Bethesda, Md.

An animated film for the anatomy, physiology and some pathology of the extraocular muscles.

This film demonstrates the possibilities of animated movies for teaching purposes and demonstrations. It was made for the teaching of flight surgeons.

FILM NO. 7
Recession of Overacting Inferior Oblique in Superior Rectus Paresis
F. BRUCE FRALICK, M.D.
Ann Arbor, Mich.

Recession of the inferior oblique in association with paresis of the contralateral superior rectus has largely replaced myomectomy of the inferior oblique in our clinic, because of the greater control over the effect obtained by recession. This operation is more easily performed in combination with resection of the externus.

The movie demonstrates our interpretation of James W. White's operation, both with and without resection of the externus.

Motion Pictures, Otolaryngology

WEDNESDAY, OCT. 11, 1944

2 p.m.

GRAND BALL ROOM

Presiding Officer:
CHEVALIER L. JACKSON, M.D.
Third Vice President

Secretary:
O. E. VAN ALYEA, M.D.

FILM NO. 1
Anatomy of the Ear
As Seen by the Otologic Surgeon
SAMUEL J. KOPETZKY, M.D.
New York, N. Y.
and
RALPH ALMOUR, M.D.
New York, N. Y.

This movie shows the anatomy of the ear as the surgeon sees it. It is not an

abstract picture of anatomical structures. The important land-marks in their relation to diverse surgical procedures are each presented and their significance stressed.

The guides toward safety in surgery are shown. Age changes in the temporal bone which vary the relationship of one vital structure to the other are demonstrated. Prevalent variations from the normal are shown, with particular reference to their recognition before surgical accidents happen. Actual anatomical sections, diagramatic animations and basic anatomical pictures form the film.

FILM NO. 2
Seventh Nerve Pathology
JOHN L. MYERS, M.D.
Kansas City, Mo.

Study of seventh nerve and collateral connections, illustrated with anatomical pictures and demonstrated by motion pictures of cases.

FILM NO. 3
The Treatment of Laryngeal and Tracheal Stenosis
GORDON B. NEW, M.D.
Rochester, Minn.

There are three stages in the treatment of laryngeal and tracheal strictures requiring skin grafting. In addition, it may be necessary first to lower the tracheotomy tube well below the stricture.

Stage one: Excision of the scarring and skin grafting.

Stage two: Maintaining the recently made airway with a prosthesis.

Stage three: Closure of the tracheotomy opening.

The movie illustrates these three stages by means of cases and drawings.

FILM NO. 4
The Pushback Cleft Palate Operation
GORDON B. NEW, M.D.
Rochester, Minn.

The movie shows the technic of the one-stage pushback operation for cleft of the hard and soft palate. There are several drawings which aid to illustrate the method. It has been found to be a valuable procedure in the care of postoperative short palates and partial clefts of the hard and soft palate.

FILM NO. 5
Kodachrome Cinemetography of the Epipharynx
S. EUGENE DALTON, M.D.
Atlantic City, N. J.

First — The method used in photographing the epipharynx.

Second — Anatomy of the epipharynx

Third — Epipharyngeal tumors

Fourth — Determining factors producing overgrowth of lymphoid tissue in the epipharynx. Visual results of experiments on representative cases showing the effect of thyroid gland dysfunction and paranasal sinus infection on this overgrowth, its prevention and eradication.

FILM NO. 6
Muscles of Phonation and Deglutition As Viewed Through Operative Wound of the Frontal and Ethmoid Sinuses
W. LIKELY SIMPSON, M.D.
Memphis, Tenn.

A large nonmalignant tumor of the frontal, ethmoid and sphenoid region was removed, leaving a large opening in this region. Through this opening a movie was made showing the action of the muscles of the soft palate, eustachian tubes and walls of the nasopharynx and the upper part of the hypopharynx.

FILM NO. 7
Fenestra Nov-Ovalis Operation for the Improvement of Hearing in Clinical Otosclerosis
JULIUS LEMPERT, M.D.
New York, N. Y.

A ten reel kodachrome motion picture demonstration of the Fenestra Nov-Ovalis Technic made with the Lempert Lightoscope, which for the first time permits visualization of the minutest anatomical structures involved in this surgery.

FILM NO. 8
Benign and Malignant Tumors of the Larynx
GABRIEL TUCKER, M.D.
Philadelphia, Pa.

The film shown also covers "Inflammatory Tumors" and will demonstrate the method of surgical treatment of both benign and malignant growths.

Motion Pictures, Joint Session

THURSDAY, OCTOBER 12, 1944

2 p.m.

GRAND BALL ROOM

Presiding Officer:

HAROLD F. WHALMAN, M.D.
Second Vice President

Secretary:

ALGERNON B. REESE, M.D.

FILM NO. 1
The Right to Hear
Committee on the Conservation of Hearing

The film is sponsored by the Committee on the Conservation of Hearing of the American Academy of Ophthalmology and Otolaryngology, and completed by the Bureau of Visual Instruction, Extension Division, State University of Iowa, Iowa City. The film was produced under the direction of the Department of Otolaryngology and Oral Surgery of the University of Iowa College of Medicine with the cooperation of the Department of Speech.

The picture dramatizes the need for organized programs of conservation of hearing and emphasizes the objectives and possibilities of attainment in a carefully planned approach to the problems involved. The technics of group and individual hearing tests, medical follow-up procedures, and educational readjustments are demonstrated. Many of the details for organizing a conservation of hearing program are suggested in the film.

FILM NO. 2
Histaminic Cephalgia
BAYARD T. HORTON, M.D.
Rochester, Minnesota
BY INVITATION

Histaminic cephalgia is a specific unilateral type of headache which has recently been recognized as a new and distinct clinical entity. The attacks are of short duration. Generally, they last less than one hour, commence and often terminate suddenly, and tend to awaken the patient at night, one to two hours after he has gone to sleep. The pain is invariably so severe that he frequently jumps out of bed before he is fully awake. Pain is the outstanding complaint and is of a constant, excruciating, burning and boring character. It involves the region of the eye, the temples, the neck and often the face and may even extend into the shoulder on the involved side. It also may spread into the upper teeth and occasionally into the lower teeth. The disease often begins after middle age.

It is also associated with the following signs, all of which appear on the affected side: profuse watering and congestion of the eye; rhinorrhea or stuffiness of the nostril; increased perspiration and frequently evidence of vasodilatation. Swelling of the temporal vessels may be noted. So severe and frequent are the attacks of pain — as often as every two hours, day and night, for a period of months — that almost every patient has contemplated suicide.

During and following the attacks, marked tenderness is frequently found when pressure is applied over the branches of the external and common carotid arteries. The pain is not confined to the distribution of any cranial nerve but has a tendency to conform to the ramifications of the external carotid artery. Most of the patients with histaminic cephalgia had had numerous operations without relief. They had been incapacitated by the disorder and had found no relief from the usual methods of treatment.

INDUCED ATTACK

The subcutaneous injection of 0.35 mg. of histamine base will precipitate an attack in the average patient with this disease. Physicians, in carrying out provocative tests, must differentiate clearly between the immediate generalized headache which so frequently results from the subcutaneous injection of 0.35 mg. of histamine base and the unilateral histaminic cephalgia which may develop thirty to fifty minutes later. The patient is unable to distinguish between induced and spontaneous attacks, as the two are identical. It is not possible to induce attacks of this character by giving histamine to normal persons. Both types of attacks may be terminated promptly by giving epinephrine intravenously (1:400,000 dilution).

DIFFERENTIAL DIAGNOSIS

Histaminic cephalgia is not to be confused with trigeminal neuralgia because trigger zones are invariably absent in histaminic cephalgia and the pain does not follow the distribution of the fifth nerve. It should not be confused with migraine

because nausea, vomiting and scotomata are invariably absent in histaminic cephalgia, and the attack usually lasts less than an hour. Heredity plays no role in histaminic cephalgia.

Unilateral recurrent erosion of the cornea which occurs at night or on opening the involved eye in the early morning may be mistaken for histaminic cephalgia. Pain is confined to the orbit in recurrent ulcer of the cornea, but not with histaminic cephalgia. Blepharospasm and photophobia occur with recurrent erosion of the cornea, but not with histaminic cephalgia. Furthermore, an examination of the cornea with the slit lamp will reveal erosions if the patient is examined during the attack. However, such erosions heal promptly, and if the patient is examined, even a few hours later, there may be no evidence of such lesions.

TREATMENT

The object of treatment is, first, to free the patient from pain, which can be accomplished without much difficulty by means of histamine desensitization. Treatment is given according to the following schedule: The contents of a 1 c.c. ampule of histamine diphosphate (0.275 mg. of histamine diphosphate) is the equivalent to 0.1 mg. of histamine base. Ampules of this character are now on the market. All doses are given subcutaneously. The quantity given at each injection is as follows: first injection, 0.10 c.c.; second, 0.15 c.c.; third, 0.20 c.c.; fourth, 0.25 c.c.; fifth, 0.30 c.c.; sixth, 0.35 c.c.; seventh, 0.40 c.c.; eighth, 0.45 c.c.; ninth, 0.50 c.c. When this type of treatment was first begun, somewhat larger doses were employed, but it was found that the smaller ones are just as effective, although in some instances the dose is built up to 1 c.c. [See **Journal of the American Medical Association,** vol. 116, pp. 377-383 (Feb. 1) 1941.] We have never given more than 1 c.c. of this solution at any time.

If, at any time, the patient notices slight flushing of the face or any other symptoms indicative of subjective or objective response to the drug, the next dose should be reduced 50 per cent. An attempt then is made gradually to increase the dose again.

These increasing doses are injected subcutaneously twice daily for approximately ten days to three weeks. After the patient is free from attacks, the second phase of the problem is to prevent future attacks. This can best be accomplished by giving each patient a proper maintenance dose. This will consist in giving the patient approximately 0.50 c.c. of histamine diphosphate (1 c.c. contains 0.275 mg. of histamine diphosphate or 0.1 mg. of histamine base) one to three times weekly, whereas a few patients apparently do not require a maintenance dose.

FILM NO. 3
Restoration of Obliterated Eye Socket
RAMON CASTROVIEJO, M.D.
New York, N. Y.

This operation is similar to the one advocated by Dr. John M. Wheeler, with the only difference that before lining the socket with the epithelial graft, a gold sphere is implanted, to give better support to the prosthesis.

FILM NO. 4
Four Operations for Glaucoma
RAMON CASTROVIEJO, M.D.
New York, N. Y.

These films illustrate the technic and postoperative results of Elliot's sclerectomy, modified LaGrange sclerectomy, iridencleisis, and cyclodialysis.

FILM NO. 5
The Marcus-Gunn Jaw-Winking Reflex
EDMUND B. SPAETH, M.D.
Philadelphia, Pa.

A presentation of 2 cases of the jaw-winking reflex as described by Marcus-Gunn (1883), one bilateral in type and accompanied by other oculomotor signs of developmental defects in the periaxial mesoderm from which the ocular muscles normally develop. The isolated integrity of the levators in this case illustrates rather well the early age in intrauterine life at which this reflex develops. While it neither proves nor denys the contention of Grant, Lewy, and Groff that the locus for the reflex lies with its efferent pathway via autonomic fibers through the ophthalmic division of the trigeminal nerve it suggests very strongly that the phenomenon can be explained on the basis of neuronal intercommunications between the trigeminal and oculomotor (and perhaps the facial) nerves. In this case, both the opening and closure of the jaw elicited the lid reflex, though to dissimilar degrees. The second case is one of unilateral jaw-closure lid-elevation.

The surgery for the 2 cases is illustrated but because of the other oculomotor complications in the first of the 2, the second alone is to be considered as descriptive of surgical technic. The surgical technic is the same as that outlined several years ago for the pseudo-Graefe syndrome of misdirected regenerating third nerve fibers.

FILM NO. 6
Catgut Sutures for Closure of the Deep Corneoscleral Wound After Cataract Extraction

FREDERICK ALLISON DAVIS, M.D.
Madison, Wis.

This film depicts a new subconjunctival buried catgut stitch for closure of the deep corneoscleral wound after cataract extraction.

The Graefe knife-scissors and the keratome scissors technic recently described and advocated by a number of surgeons have been employed in these operations.

The catgut stitch consists of one and, at times, two plain five-0 catgut sutures which are inserted immediately after the chamber is opened, before enlargement of the incision with scissors. The catgut is attached to a fine, Kalt-type corneal needle. Closure of the incision is accomplished by a simple appositional suture inserted in the outer margins of the cornea and sclera, which produces very satisfactory coaptation of the lips of the wound. A conjunctival flap is sutured separately with five to seven fine silk sutures and thereby made to cover completely the deep corneoscleral incision.

The extractions are by the intracapsular method previously described and shown in this Section by the author.

A more detailed description of the operative technic used in this film will be found in a brief article published by the author in the April, 1944, issue of the Archives of Ophthalmology.

FILM NO. 7
A New Surgical Procedure for Congenital Cataract

WILLIAM F. MONCREIFF, M.D.
Chicago, Ill.

This film depicts a surgical procedure for congenital cataract involving a new principle (not extraction) by means of which a rapid and complete result is obtained with a single operation.

An absolutely essential preoperative condition is dilatation of the pupil **ad maximum.** The operation comprises two steps, the first being discission, in which the first incision into the lens is along the full vertical diameter of the pupil from below upward, followed by multiple incisions in all meridians of the lens, thus creating a large central opening in the anterior capsule and reducing the lens nucleus and those cortical fibers within the pupillary zone to small fragments which will float freely in the aqueous. A special discission knife is used, its blade almost 3 mm. long, and wide enough to make a limbal incision which just admits the cannula of the (Goldstein) irrigator. The second step is the displacement of these lens fragments forward into the anterior chamber by the use of the anterior chamber irrigator.

After partial emptying of the anterior chamber, the tip of the irrigator cannula is introduced through the incision, fluid is injected, and the finely divided particles of lens substance come forward in clouds into the anterior chamber.

SECTION ON INSTRUCTION

Officers

DEAN M. LIERLE, M.D.
Secretary for Instruction — Otolaryngology
A. D. RUEDEMANN, M.D.
Secretary for Instruction — Ophthalmology

THIS SECTION PRESENTS FOR 1944:

1. Thirty-six individual and nineteen sequential ophthalmological courses with a faculty of approximately seventy-five instructors covering one hundred seventy-one hours.
2. Thirty-two individual and thirteen sequential otolaryngological courses with a faculty of forty-four instructors and covering one hundred twenty-seven hours.

Note

Last year the length of the instructional period was reduced from one and one-half to one hour and there were three instead of two periods each morning. More continuous courses were presented because some of the subjects could not be adequately covered in one period. The arrangement has proved to be very satisfactory and it will be used again this year.

The first instructional period begins at 8:30 a.m. and it is urged that each one be in his seat at least ten minutes before the conference starts. The attendants at the doors are instructed not to admit anyone to the instructional courses without a ticket. This is necessary because the seating capacity is limited and only a definite number of tickets can be sold for each course. In the event of loss of a ticket, please report to Room 701 at the Palmer House.

There will be a better choice of courses if tickets are contracted for in advance, and it would also eliminate considerable confusion during the registration.

The Secretaries of Instruction would appreciate any suggestions regarding subjects for courses and instructors and any other criticisms which will be of value in improving the courses.

General Information

Conference Periods

MONDAY, TUESDAY AND WEDNESDAY

MORNINGS

($1.50 for each period of instruction)
First Period.....8:30 a.m. to 9:30 a.m.
Second Period.10:00 a.m. to 11:00 a.m.
Third Period..11:30 a.m. to 12:30 p.m.

SEVENTH FLOOR — PALMER HOUSE

The morning periods will be designated with the following key:

M-1.....Monday, First Period 8:30 a.m. to 9:30 a.m.
M-2.....Monday, Second Period 10:00 a.m. to 11:00 a.m.
M-3.....Monday, Third Period 11:30 a.m. to 12:30 p.m.
T-1.....Tuesday, First Period 8:30 a.m. to 9:30 a.m.
T-2.....Tuesday, Second Period 10:00 a.m. to 11:00 a.m.
T-3.....Tuesday, Third Period 11:30 a.m. to 12:30 p.m.
W-1.....Wednesday, First Period 8:30 a.m. to 9:30 a.m.
W-2.....Wednesday, Second Period 10:00 a.m. to 11:00 a.m.
W-3.....Wednesday, Third Period 11:30 a.m. to 12:30 p.m.

(Familiarity with this key will be helpful and will avoid confusion)

General Instructions

Members only who have paid their 1944 dues may make reservations in advance of the meeting. After the session opens no preference will be given to members that is not extended to candidates and guests.

Orders unaccompanied by properly executed checks will not be filled.

Refunds will be granted only if request for reimbursements is received prior to October 5.

Academy candidates *whose names appear in this issue of the* TRANSACTIONS are exempt from paying registration fee; however, even they are not privileged to reserve tickets in advance of October 8.

Candidates *whose names do not appear in this* TRANSACTIONS and guests will be required to pay a registration fee of five dollars ($5.00).

All orders for tickets will be filled according to postmark on envelope. Previous to October 1, send orders to 100 First Avenue Building, Rochester, Minnesota; after October 1, mail orders direct to Dr. William L. Benedict, c/o Palmer House, Chicago, Illinois.

Requesting Conference Tickets

At the end of this section is printed a colored sheet to be used when ordering tickets in advance. Specify preferences on this sheet and mail to the executive office. Careful following of instructions will insure the correct filling of your requests. *Since courses are limited in number it is necessary that you select more courses than it is possible for you to attend; in other words, a list of three preferences for each period for each day should be given.* Please note carefully that where continuous courses are selected the additional periods open in your schedule are varied.

If you are requesting registration in the continuous courses, please specify preferences for individual conferences as substitutes in order to avoid disappointment should the continuous courses which you select be sold out when your order is received.

Claiming Conference Tickets

There will be an *Advance Order Registration Section* where members who have sent advance orders will register and secure conference tickets.

Please Note: Only members in good standing will be accommodated in this way. Members delinquent in dues must make selection of courses in Chicago.

Synopsis of Courses

Synopses of courses for which tickets are purchased will be supplied. Please check your assignments at time of registration to see that the proper synopses are in your envelope. As usual, monographs and sets of synopses covering courses presented may be purchased at the General Registration Desk.

PROGRAM OF COURSES

Continuous Courses

OPHTHALMOLOGY

1944

COURSE 1

Room 752 Periods M-1, T-1, W-1

ARTHUR J. BEDELL, M.D.
Albany, N. Y.

Medical Ophthalmoscopy
A three period course — $4.50

Limited to 40

This will be a continuation of former courses on fundus changes found in constitutional diseases; their significance in diagnosis, prognosis and treatment.

New cases will be illustrated with kodachrome photographs. The common alterations as well as the rare ones will be presented.

COURSE 2

Room 754 Periods M-3, T-3, W-3

JOHN H. DUNNINGTON, M.D.
New York, N. Y.

Surgery of the Eyelids and Orbit
A three period course — $4.50

Limited to 40

The surgical correction of lacerations, particularly those involving the lid margin and the region of the inner canthus, will be considered, as well as ectropion, coloboma and other anomalies of the lids. Particular emphasis will be placed upon the operative technic and postoperative treatment of various plastic procedures used in the reconstruction of the eyelids.

The surgery of the orbit will include a discussion of (1) methods of approach for orbital neoplasms, (2) technic of orbital implantation for the correction of sunken orbit, (3) methods of restoration of contracted socket.

COURSE 3

Room 781 Periods M 1 & 2, T 1 & 2

BRUCE FRALICK, M.D.
Ann Arbor, Mich.

ELIZABETH CROSBY, M.D.
Ann Arbor, Mich.

BY INVITATION

Anatomy of the Orbit

A four period course — $6.00

Limited to 30

Understanding the anatomy of the eye is a great help to any ophthalmic surgeon in performing the various surgical procedures on the globe and adnexa. It familiarizes one with normal relationships, thereby giving confidence when conducting corrective operations in complicated cases. A knowledge of anatomy also includes an understanding of the variations from the normal and the effect these variations play on the functioning of the part. This aids the surgeon in encountering anomalous relationships.

A brief description will be given of the anatomical characteristics of the bony orbit and its contents, the eyelids, the lacrimal apparatus and nervous pathways to illustrate the various surgical problems which each tissue presents to the ophthalmic surgeon. Practical illustrations will be used throughout in an attempt to demonstrate the value of a thorough knowledge of applied ophthalmic anatomy if one is to get the highest percentage of good surgical results.

COURSE 4

Room 711 Periods M-1, T-1, W-1

WATSON GAILEY, M.D.
Bloomington, Ill.
and
E. C. ELLETT, M.D.
Memphis, Tenn.
and
D. B. KIRBY, M.D.
New York, N. Y.

Cataract

A three period course — $4.50

Limited to 35

M-1—Preparation of the Patient
Watson Gailey, M.D.

The lecture will include the preparation of the prospective surgical cataract patient from the time of the first examination in the office until the section is made.

The lecturer will particularly stress the methods used by himself but will also deal with those used by other surgeons.

T-1—Selection of Cases
E. C. Ellett, M.D.

The basis for the selection of a case for cataract operation can be considered under two heads: the eye in question, and the general health and condition of the patient.

Local conditions to be considered are the state of vision in the affected eye, and the state of vision in the fellow eye. In general it is not well to operate on an eye when the fellow eye has useful vision. The visual field of the affected eye, especially the central field, should be investigated. The tension, the lacrimal apparatus, and the condition of the conjunctiva, especially its bacteriology, should be investigated.

The general health, especially blood pressure, blood sugar, urine, teeth and lungs, all are closely related to the success of a cataract operation. The patient should be free of acute illness and not have fever. The bladder, especially with reference to the ability of the patient to urinate lying down, should be investigated.

W-1—Complications and Their Treatment
D. B. Kirby, M.D.

The general and local complications arising before, during and after operation for cataract and their prevention and handling are reviewed and discussed. The ma-

terial used and the points raised are those which have been used in lectures on the subject. In general, the application is made in relation to my own custom and practice.

The important complications — hemorrhage, glaucoma, prolapse of the iris, incarceration of the iris, presentation of vitreous, loss of vitreous, inflammation, infection, imperfect healing of the incision and corneal opacity — are treated in detail. The only conclusion possible is that if by study, by observation of the experience of others, and by analysis of his own experience, a surgeon broadens his knowledge of the prevention of these complications, he will encounter fewer of them; and if he increases his armamentarium for the handling of complications as they arise, his results will be better.

COURSE 5

Room 796

Periods M-1,2,3, T-1,2,3, W-1,2,3

H. S. GRADLE, M.D., et al.

GLAUCOMA

A nine period course — $13.50

Limited to 40

M-1—Definition, Mechanisms and Classification

Peter C. Kronfeld, M.D.
Chicago, Ill.

This paper deals with the definition, mechanisms, and classification of the ocular diseases comprised under the term "the glaucomas," associated with or due to elevated intraocular pressure. The three important factors of inflow of aqueous, outflow of aqueous, and intraocular blood volume are discussed in relation to hypertension, as well as the mechanisms influencing each. A tentative classification of the glaucomas, based upon the present knowledge, is suggested and elaborated upon.

M-2—The Effect of Pressure Upon Retina and Optic Nerve. Principles of Non-surgical Treatment of Hypertension.

Glen W. Nethercut, M.D.
Chicago, Ill.

In the first part of the lecture, the effects of elevated intraocular pressure upon the retina and optic nerve are discussed, from an anatomical as well as from a functional standpoint. The two aspects are then correlated. In the second part, the principles and methods of non-surgical treatment of elevated intraocular pressure are described at length. The lecture closes with a detailed description and discussion of methods of use of the various medications, both local and systemic, that are employed.

M-3—Principles of Surgical Treatment of Hypertension.

Harry S. Gradle, M.D.
Chicago, Ill.

This is a discussion of the surgical principles that are involved in the relief of elevated intraocular pressure. The analogy of a closed kitchen sink is used to illustrate the relationships in the anterior chamber. The various forms of drainage of the aqueous that may be employed are discussed and indications laid down for each.

T-1—Narrow Angle Glaucoma

Thomas D. Allen, M.D.
Chicago, Ill.

In certain cases of elevated intraocular pressure, the angle of the anterior chamber is very shallow and this is associated with definite clinical pictures. In the majority of cases, acute congestion of the eyeball accompanies and complicates the condition, although in some cases that phase is lacking. The clinical appearance of both conditions is described and discussed at length as well as the therapeutic measures indicated.

T-2—Diagnostic and Differential Diagnostic Problems.

Frederick C. Cordes, M.D.
San Francisco, Calif.

Before the management of a case of glaucoma may be undertaken, it is essential that it be placed in one of the accepted classifications where the etiological aspect is considered. For that reason, the author deals at length with the differential diagnostic problems of the various forms of elevated intraocular pressure and points out in detail the various aspects that allow of an accurate diagnosis which in turn indicates the proper course of treatment necessary to control the condition.

T-3—Congenital Hydrophthalmus

Ralph I. Lloyd, M.D.
Brooklyn, N. Y.

The hour is devoted to a discussion of the various aspects of congenital glaucoma, grouped under the term "hydrophthalmus." The clinical picture of each

form is discussed at length, together with statistics of incidence. Treatment is elaborated upon as is the prognosis. A complete bibliography is appended.

W-1—Glaucoma Secondary to Uveitis

F. Bruce Fralick, M.D.
Ann Arbor, Mich.

This is an elaborated discussion of hypertension due to inflammations of the uvea. There are considered the sudden pressure increases due to acute and chronic uveitis and the gradual pressure increases due to these causes. Methods of treatment are discussed, both medical and surgical.

W-2—Wide Angle or Compensated Glaucoma

Samuel J. Meyer, M.D.
Chicago, Ill.

This is the most common form of glaucoma and the one that in its earliest stages requires the most careful diagnostic acumen. The condition is described in detail, structure by structure, and the diagnostic phases are evaluated for the various stages of the disease. Provocative methods are elaborated upon for both suspected as well as existing hypertension. The over-all picture is then rounded out.

W-3—The Histopathology of Acute Glaucoma

Brittain F. Payne, Maj., M.C.
Randolph Field, Texas

Discussion of the microscopic findings of the different ocular tissues under the influence of sudden and acute increase of intraocular pressure. The variations in size and content of the various parts of the eye have a definite relationship to the clinical picture of the disease and these are correlated by the author. The lack of specimens of fresh acute hypertension leaves many gaps in the pathological picture.

W-3—The Histopathology of Chronic Glaucoma

Georgiana Dvorak-Theobald, M.D.
Oak Park, Ill.

The microscopic anatomy of the all-important chamber angle is described in detail and is correlated with the clinical picture of that type of glaucoma that is not associated with congestion. Of especial importance is the relationship between the Canal of Schlemm and the trabeculum. Due to the lack of specimens of early cases, some of the gaps in the histological picture have to be filled by deductions and speculations.

COURSE 6

Room 727 Periods T 2 & 3

LeGRAND HARDY, M.D.
and
GERTRUDE RAND, Ph.D.
BY INVITATION
New York, N. Y.

Color Vision
Recent Developments in
Color Vision Testing
A two period course — $3.00

Limited to 30

COURSE 7

Room 727 Periods W 2 & 3

LeGRAND HARDY, M.D.
and
GERTRUDE RAND, Ph.D.
BY INVITATION
New York, N. Y.

Color Vision
Recent Developments in
Color Vision Testing
A two period course — $3.00

Limited to 30

Dr. Hardy's two hour course "Color Vision" is being presented twice as Course 6 and Course 7. **Please select course number according to day on which you wish to attend.**

Color vision testing requires standardization of two out of three variables involved in the process. The most important factors in color vision testing will be outlined. The important triads in seeing color, in illumination, in the color vision process and in the characteristics of the object will be described and related to the ICI-Coordinate System and Standard Observer. Color tests will be described, evaluated and demonstrated. Recent progress in research will be discussed.

COURSE 8

Room 777

Periods M 1 & 2, T 1 & 2, W 1 & 2
LeGRAND H. HARDY, M.D., et al.

Orthoptics
A six period course — $9.00

Limited to 35

M-1 & 2—Orthoptics

LeGrand H. Hardy, M.D.
New York, N. Y.

1. Definition of orthoptics.
2. Orthoptic training and surgery.
3. Misapplications.
4. The safe approach to orthoptics.
5. Orthoptics and the heterophorias.
6. Subjective versus objective cures.
7. Orthoptics and tropias.
8. Dangers and safeguards.
9. The American Orthoptic Council.

T-1 & 2—Management of Orthoptic Therapy

Truman L. Boyes, M.D.
New York, N. Y.

An abbreviated history of orthoptics and the practical discussion of clinic organization and associated home treatment will be given. The routine management of visual and orthoptic therapy in children and adults will be discussed. There will be an analysis of statistics showing the final results and benefits between patients who had orthoptic therapy and those who had no orthoptic therapy.

W-1 & 2—The Value of Orthoptics in Private Practice

George P. Guibor, M.D.
Chicago, Ill.

An orthoptic routine should consist of a diagnostic and therapeutic procedure and of these two, the former is the more important.

In the diagnostic routine, the screen or cover test is considered the most valuable procedure used in determining the presence or absence of a motility defect. This test, therefore, will be discussed in detail.

Following the diagnosis of the type of deviation and the determination of the etiology, the therapeutic routine may include the correction of refractive errors, the use of prisms, cycloplegic agents, occlusion and fusion exercises with or without surgery. The cause of the deviation found in heterophorias or heterotropias should be determined if possible because the etiology in such a case often suggests the treatment to be employed.

Case histories of the types of strabismus seen by the oculist in infants, in youths and in older patients will be discussed from a neurologic standpoint.

COURSE 9

Room 734 **Periods M-1, T-1**

WENDELL L. HUGHES, M.D.
Hempstead, N. Y.

Lid Reconstruction
A two period course — $3.00

Limited to 35

The first hour will be devoted to surgical reconstruction of the lower lid when it is completely absent or its removal is necessitated by a malignant growth.

The second hour will be devoted to reconstruction of part of the lower lid or of the upper lid and also to total reconstruction of the upper lid.

These lectures will be illustrated by kodachrome slides and movies.

COURSE 10

Room 722 **Periods T 1 & 2**

WALTER B. LANCASTER, M.D.
Boston, Mass.
and
JULIA LANCASTER
San Francisco, Calif.
BY INVITATION

Anomalous Retinal Correspondence
A two period course — $3.00

Limited to 40

COURSE 11

Room 744 **Periods W 1 & 3**

WALTER B. LANCASTER, M.D.
Boston, Mass.
and
JULIA LANCASTER
San Francisco, Calif.
BY INVITATION

Anomalous Retinal Correspondence
A two period course — $3.00

Limited to 40

Dr. Lancaster's two hour course "Anomalous Retinal Correspondence" is being presented twice as Course 10 and Course 11. **Please select course number according to day on which you wish to attend.**

Normal retinal correspondence will be defined and described. The conditions under which anomalous correspondence is substituted as a working condition will be discussed; how it works, what good it does (avoids diplopia) and what harm

it does (makes binocular vision in the regular normal way impossible). Tests such as major amblyoscope, double image test and after image test for detecting anomalous correspondence will be described. Technic: its difficulties, advantages and disadvantages and its bearing on treatment and prognosis will be presented. How anomalous correspondence affects the results of operation will be shown.

Orthoptist, Miss Lancaster, will demonstrate the technic of diagnosis and also of treatment on actual patients. Home treatment, its importance and its pitfalls will be described. The correction of anomalous correspondence establishes true diplopia. The fusion of the true images is of great value in the correction of squint, whether postoperatively or without surgery. It makes fusion training with simple or no equipment possible which is especially valuable in home training. Fusion of anomalous images does not correct squint.

COURSE 12

Room 733

Periods M 2 & 3, T 2 & 3, W 2 & 3

DONALD J. LYLE, M.D.
Cincinnati, Ohio

Neuro-Ophthalmology
A six period course — $9.00

Limited to 35

This course will include a discussion of the anatomy and physiology of the central nervous system with pathological application in reference to ophthalmology.

A description will be given of the visual system from the retina through the conductive paths to the visual, sensory and psychic areas of the brain.

The voluntary and autonomic motor system of the eye will be discussed.

Special attention will be given to a discussion of reflexes and syndromes concerned with ophthalmology.

COURSE 13

Room 712

Periods M 2 & 3, T 2 & 3, W 2 & 3

ROBERT J. MASTERS, M.D.
Indianapolis, Ind.
HARVEY E. THORPE, M.D.
Pittsburgh, Pa.
PETER C. KRONFELD, M.D.
Chicago, Ill.

Slit Lamp Biomicroscopy
A six period course — $9.00

Limited to 30

The lectures and demonstrations of this course are planned for beginners in slit-lamp microscopy and for those registrants who desire a review of the elementary features of biomicroscopy.

The early part of each session is devoted to an illustrated lecture, which is followed by a period of actual work with the slit lamps by the members of the class.

The illustrated lectures cover, in regular order from day to day, studies of the normal and pathological cornea, anterior chamber, iris, crystalline lens, and vitreous humor. Special attention is given to the findings which can be made with no other instrument, and which make the slit lamp indispensable in the routine practice of ophthalmology.

The demonstration period is utilized to make each class member familiar with the manipulation of the instruments. During each demonstration period a round table, question-and-answer discussion is carried on for the class members who are already familiar with the instruments.

COURSE 14

Room 705

Periods M 2 & 3, T 2 & 3, W 2 & 3

BRITTAIN F. PAYNE, MAJOR, M.C.
et al.

Ophthalmology in Aviation
A six period course — $9.00

Limited to 35

M-2—Visual Standards and Their Relationship to Problems in Combat Aviation

V. A. Byrnes, Col., M.C.
Randolph Field, Texas

The various factors of depth perception, visual acuity, accommodation, muscle balance and color vision are related to specific tasks in combat flying. The use of these factors in landing, in maintaining position in formation, on bombing runs, in evasive action and in combat firing is discussed as well as the exacting nature of these visual tasks.

M-3—The Use of Corrective, Colored and Protective Lenses in Aviation

John L. Matthews, Lt. Col., M.C.
Randolph Field, Texas

The especial problems of the Flight Surgeon are discussed in prescribing refractive corrections for AAF personnel operating under various flying conditions, in furnishing protective lenses and goggles for use in aircraft, and in providing colored lenses to meet the problems of glare arising at altitudes, over water, and in the Arctic and desert.

T-2—Effect of Climate Upon the Eyes in Various Combat Zones

Merrill J. Reeh, Col., M.C.
Randolph Field, Texas

Pilots and combat crews live and fly under every climatic condition from the equator to the poles. Flights are frequently of long duration and at high altitudes, over water, mountains, jungles and desert. The various climatic conditions, coupled with fatigue, cold and anoxia, produce numerous effects upon the eyes.

T-3—The Treatment of Ocular Diseases with Penicillin and Its Adjunctives

Richard G. Scobee, Lt., M.C.
Randolph Field, Texas

Specific consideration will be given to orbital cellulitis, endophthalmitis, conjunctivitis, infected corneal ulcers and uveitis. Particular attention will be paid to uveitis, since previous therapeutic regimens have been relatively unsatisfactory in this condition. The various routes of administration of penicillin, together with the effective blood levels attained with each, will be considered. When adjuncts to penicillin therapy are indicated, these will be mentioned.

W-2—The Care of Ocular Injuries Returned from Various War Theaters

Brittain F. Payne, Major, M.C.
Randolph Field, Texas

Serious ocular injuries are often evacuated by air from the South Pacific, European and Far Eastern war areas. Many of the patients are sent to Brooke General Hospital, San Antonio, Texas. Lacerations, burns, intraocular missiles, detachments of the retina, intraocular hemorrhages, traumatic iridocyclitis and ocular complications from tropical disease are the types of cases seen. The treatment involves complete laboratory examinations with adequate surgical and medical treatment.

W-3—Problems of Night Vision in Combat Aviation

V. A. Byrnes, Col., M.C.
Randolph Field, Texas

This presentation will cover briefly the amount of illumination in the night sky, problems of adapting fighter and bomber pilots for night flights, the effect of anoxia on night vision, problems in illumination of cockpit, instruments, maps and charts and equipment used, and visibility of lights to the enemy. There will be a discussion of tests used for night vision determinations.

COURSE 15

Room 738
Periods M-1, 2, 3; T-1, 2, 3; W-1, 2, 3
A. D. PRANGEN, M.D., et al.

REFRACTION

A nine period course — $13.50

Limited to 45

M 1 & 2—Manifest Refraction — Evaluation of the Patient

Hendrie W. Grant, M.D.
St. Paul, Minn.

Refraction is an integral part of the complete ophthalmological examination. Certain symptoms have been known as those which produce asthenopia or fatigue. These symptoms and their relation to the habits, occupation and physical and nervous make-up of the individual are carefully evaluated. The method of manifest refraction with the cross cylinder is demonstrated, keeping constantly in mind the importance of the sphere.

The mechanism of accommodation is brought about by a contraction of the ciliary body with a resulting increase in the convexity of the lens. The amplitude reaches its peak in the early teens and gradually decreases with age. Amplitude of accommodation has a wide normal variation which must be constantly born in mind. Presbyopia is due to age changes in the ciliary body and sclerosis of the lens nucleus. Subnormal accommodation is due to a weakness of the ciliary body from toxic effects on the ciliary nerves or muscle. Illustrative cases are shown.

Converging power is best measured by the method of Duane, computed from the

convergent near point and the intracentral base line. Exophoria may be the cause of symptoms of fatigue but does not of itself produce a remoteness of the convergent near point. Remoteness of the convergent near point is due to a functional or organic disturbance of the convergent center. Illustrative cases of the above with the treatment are demonstrated.

M-3—Retinoscopy and Crossed Cylinders

D. H. O'Rourke, M.D., Denver, Colo.

Skiascopy (retinoscopy) is the most accurate objective method of determining the refractive error of the eye. Only spherocylindric skiascopy with the plane mirror will be considered. The Copeland Refractascope has several advantages both mechanically and scientifically, and its use will be demonstrated.

The crossed cylinder is a most valuable method for the subjective testing of the refractive error of the eye. This simple device used intelligently affords positive information in regard to strength of cylinder and axis of cylinder. Demonstration of its use rather than the written word makes its clinical use easily understood.

T-1—Presbyopia

Presbyopia introduces the problems concerned with the failing or failure of the accommodative mechanism. In this and the foregoing subjects clinical application will be stressed.

T-2—Cycloplegic Examination and Refraction

A. D. Prangen, M.D.
Rochester, Minn.

Indications and contraindications for the use of cycloplegic drugs will be discussed. The several drugs, their action, methods of application and selection, will be taken up. Selection of cycloplegia and its use in different age periods will be discussed. The question as to what constitutes satisfactory and adequate cycloplegia will be discussed. Residual cycloplegia at the time of refraction and methods for its measurement will be described. The significance of full cycloplegic refractive findings and their relation to postcycloplegic findings wil be stated. Postcycloplegic examination, technic, and application to final prescription will be described.

T-3—Characteristics, Management and Prescribing for Various Refractive Types

The characteristics, differential interpretation, management and prescribing for hyperopia, myopia and astigmatism will be analyzed. Prescribing and handling of marked astigmatism, including partial correction for astigmatism will be discussed. Anisometropia, antimetropia and irregular astigmatism, their detection and correction will be described.

W 1 & 2—Refraction Without Cycloplegic

James J. Regan, M.D., Boston, Mass.

Admitting that use of a cycloplegic is desirable in children under fifteen and almost indispensable in children under nine, it will be our purpose to present a technic for measuring refractive errors subjectively, without the use of cycloplegic, which we have found dependable over a period of years, even when checked against cycloplegic examination of the same (identical) cases.

There is no hard and fast age limit. The technic is recommended generally for patients over fifteen years of age, though, on many occasions when time permitted, comparisons were made with cycloplegic studies on children under nine and the results were similar. There will, of course, be cases in age groups up to sixty which, for one reason or another, should be refracted under cycloplegic, and we highly recommend mydriasis for better ophthalmoscopy. But, for the purpose of prescribing adequate correction for the great majority of refractive errors, subjective refraction presents many advantages. If one wishes to test its reliability, one needs only to substitute this for the usual manifest test, and compare the results with cycloplegic and postcycloplegic tests.

1. The therapy and practice of fogging. The importance of controlling accommodation, particularly for the measurement of the astigmatic error. The value of astigmatic dials, and proper technic for their use. The advantage of fogging in subjective examination, even under cycloplegia.
2. Demonstration of the Lancaster technic, using members of the class (preferably those wearing corrections prescribed upon findings of a complete cycloplegic examination).

W-3—Round Table Discussion

COURSE 16

Room 722 Periods M 1 & 2

EDMUND B. SPAETH, M.D.

Philadelphia, Pa.

Immediate Treatment of Injury of the Lids with Special Reference to Type of Injury Seen in War

A two period course — $3.00

Limited to 40

COURSE 17

Room 722 Periods W 1 & 2

EDMUND B. SPAETH, M.D.

Philadelphia, Pa.

Immediate Treatment of Injury of the Lids with Special Reference to Type of Injury Seen in War

A two period course — $3.00

Limited to 40

Dr. Spaeth's two period course, "Immediate Treatment of Injury of the Lids with Special Reference to Type of Injury Seen in War," is being presented twice as Course 16 and Course 17. **Please select course number according to day on which you wish to attend.**

The immediate treatment of injuries of the lids, especially injuries from warfare, when handled as immediate surgical problems results in a marked decrease in subsequent surgery, minimizes decidedly later permanent deformity, and is frequently the means of conserving useful vision. This truism is the principle for presentation of this course.

COURSE 18

Room 734 Periods M-3, T-3, W-3

H. S. SUGAR, MAJOR, M.C.

Vancouver, Wash.

Extraocular Muscles
Diagnosis and Treatment

A three period course — $4.50

Limited to 35

This course will deal with the practical application of the principles for treating extraocular muscle anomalies. It will include what little we know of the etiology of concomitant and paretic squint, their diagnosis and principles of treatment, both orthoptic and surgical. A simple practical routine for the handling of muscle anomalies will be discussed.

COURSE 19

Room 715

Periods M-1,2,3; T-1,2,3; W-1,2,3

GEORGIANA DVORAK-THEOBALD, M.D., et al.

PATHOLOGY

A nine period course — $13.50

Limited to 25

M-1—Histology of the Human Eye

Brittain F. Payne, Maj., M.C., A.U.S.
Randolph Field, Texas

The application of histology to the routine practice of ophthalmology affords better insight into the recognition and treatment of ocular disease. This portion of the course in Histopathology of the Eye will be illustrated by sections prepared and stained by Dr. Edgar B. Burchell of the New York Eye and Ear Infirmary and used by Dr. Bernard Samuels in his lectures on this subject. Each student will have a section of a normal eye for study and the lecture will be illustrated by various specimens stained to show characteristic findings.

M-2—Acute and Subacute Inflammations.

Georgiana Dvorak-Theobald, M.D.
Oak Park, Ill.

M-3—Tuberculosis of the Eye

John M. McLean, M.D.
New York, N. Y.

T-1 & 2 — Microscopy and Photomicroscopy.

Max Poser, Ph.D.
BY INVITATION
Rochester, N. Y.

1. The lecture will include an explanation of the optical laws involved in the formation of the correct image of the object viewed through the microscope.
2. Technic of illumination of the various kinds in microscopy.
3. Dark field illumination including paraboloid and cardioid condensor studies.
4. Critical illumination, and Koehler illumination principles.
5. Polarizing microscope and its application in biology and medicine.
6. Technic in photomicroscopy.

T-3—Retinal Tumors

John M. McLean, M.D.
New York, N. Y.

W-1 & 2—Retinal Detachment

Charles A. Perera, M.D.
New York, N. Y.

The pathology of retinal detachment will be reviewed from an etiologic basis. Retinal changes, predisposing to tears in this structure with ensuing detachment, will be reviewed, and examples demonstrated. The survey of this subject will include separation of the retina as a result of contracture and hemorrhages on its internal surface, retinal detachment following the presence of fluid or solid tissue between the pigment epithelium and remaining layers of the retina, and traumatic separation of the retina.

W-3—Tumors of the Uveal Tract

Brittain F. Payne, Maj., M.C., A.U.S.
Randolph Field, Texas

The predominant tumor of the uvea is the malignant melanoma or melanosarcoma. Approximately 95 per cent of these tumors arise in the choroid, 4 per cent in the ciliary body, and less than 1 per cent in the iris.

The classification involves pigmentation or nonpigmentation (leucosarcoma); flat or elevated; globular or pedunculated; and the structure or tissue from which they arise. The stages in development will be discussed.

The anatomical location of tumors has considerable bearing on metastasis and prognosis.

COURSE 20

Room 728

Periods M 2 & 3, T 2 & 3, W 2 & 3

JAMES W. WHITE, M.D., et al.

Anomalies of the Extraocular Muscles
A six period course — $9.00

Limited to 35

M-2—Physiology and Pathology of the Extraocular Muscles

Harold W. Brown, M.D.
New York, N. Y.

BY INVITATION

The physiological actions of the ocular muscles will be discussed.

a. As individual muscles.
b. In the acts of associated movements.
c. In the disjunctive acts of convergence, divergence, sursumvergence and rotations.

From these physiological acts, the pathological variations from normal are developed and classified, and a method of differential diagnosis outlined.

M-3, T-2—Tests

Maynard C. Wheeler, M.D.
New York, N. Y.

This willi nclude a demonstartion and discussion of the most important methods used in the diagnosis and measurement of muscle defects. Particular emphasis will be placed on the screen test and its actual application in routine practice.

T-3—Congenital Anomalies

Rudolf Aebli, M.D.
New York, N. Y.

The subject matter will cover the more usual congenital anomalies such as retraction syndrome and its variations, strabismus fixus, Marcus Gunn or jaw winking syndrome, paralyses of both elevators of one eye with true and pseudoptosis and also congenital individual muscle anomalies.

W-2—The Surgical Treatment of Heterophoria and Heterotropia

John H. Dunnington, M.D.
New York, N. Y.

Special emphasis will be placed upon the importance of selecting the proper type of operative procedure. A brief review of the methods used to aid in this selection will then be given, followed by a full discussion of the indications and contraindications for the different types of surgical procedures.

W-3—Nonoperative Treatment and the Relation of Ametropia to Motor Anomalies

James W. White, M.D.
New York, N. Y.

In considering the various methods of nonsurgical treatment of the phorias and tropias, a definite differential diagnosis must be made. All treatment depends on the proper estimation of the various points in the diagnosis. Treatment will include the use or nonuse of glasses for the various types of deviations and the indications for prismatic correction of the various phorias and tropias. The principle of orthoptic training will be discussed as to the results to be expected from their use in the different types of anomalies.

COURSE 21

Room 736 **Periods M-1, T-1, W-1**

JAMES W. WHITE, M.D.

and

HAROLD W. BROWN, M.D.

BY INVITATION

New York, N. Y.

Ocular Motor Anomalies
Extension Course
A three period course — $4.50

Limited to 40

In the three periods assigned to this course, the physiological action of the extraocular muscles will be reviewed and the principles of the pathological variations developed. Tests will be considered with their uses, interpretations and limitations.

The nonsurgical and surgical treatment will be discussed.

COURSE 22

Room 706

Periods M 1 & 3, T 1 & 3, W 1 & 3

ALAN C. WOODS, M.D.

Baltimore, Md.

Medical Ophthalmology
A six period course — $9.00

Limited to 35

The lectures will cover the following subjects:

1. The clinical manifestations of ocular tuberculosis.
2. Diagnosis, pathogenesis and treatment of ocular tuberculosis.
3. Ocular complications of hyperthyroidism and hypoparathyroidism.
4. Sarcoid-uveo-parotid fever — Mikulicz's syndrome.
5. Brucellosis.
6. Focal infection.

Continuous Courses
OTOLARYNGOLOGY
1944

COURSE 50

Room 744 **Periods T-1, W-1**

LORAINE V. ANSON, M.A.

Iowa City, Iowa

BY INVITATION

Testing the Hearing of School Children
A two period course — $3.00

Limited to 40

Active cooperation between otolaryngologists and educators is essential in order that school hearing test programs culminate in effective medical and educational follow-up procedures. A school conservation of hearing program is described which places special emphasis on cooperation between the otolaryngologist and the educator, selection of the testing room, equipment, testing technics, evaluation of test results, administrative blanks and forms, and criteria for medical referral and educational readjustment.

COURSE 51

Room 706 **Periods M-2, T-2, W-2**

LOUIS H. CLERF, M.D.

Philadelphia, Pa.

Causes of Hoarseness with Particular Reference to Treatment
A three period course — $4.50

Limited to 35

This course is devoted to a brief consideration of the symptom "hoarseness" and the diagnosis of conditions producing it.

Diseases of the larynx are discussed especially from the standpoint of treatment, and many of the more common typical and atypical laryngeal lesions are shown in kodachrome with movie film.

COURSE 52

Room 712 **Periods M-1, T-1**

STUART CULLEN, M.D.

Iowa City, Iowa

BY INVITATION

Anesthesia in Otolaryngology
A two period course — $3.00

Limited to 30

The course this year has been altered by making it a two period continuous course. By so doing, it is hoped that a more complete exposition of the practical factors involved in the selection and administration of anesthesia can be made. There will be a discussion of drugs used for premedication, and specific examples will be given of the use of the drugs in different combinations and dosage according to the requirements of the anesthesia. There will be a discussion of the available agents and technics in modern anesthesia

with direct reference made to their particular applicability in otolaryngology. A detailed account will be given of the advantages and disadvantages of the application of the endotracheal technic in otolaryngological surgery. There will be more emphasis this year than in the past on the use of cocaine and drugs with similar action. This will include a description of the use of nerve blocks for diagnostic and anesthetic purposes and a thorough review of the signs and symptoms of cocaine poisoning and its specific treatment.

There will also be a discussion of the rational use of pentothal sodium in otolaryngology which will include the currently accepted technic and specific indications and contraindications for its use. A new feature will be a discussion of the use of curare to provide relaxation and facilitate endoscopy.

COURSE 53

Room 711 Periods M-3, T-3, W-3

NORMAND L. HOERR, M.D.

Cleveland, Ohio

BY INVITATION

Key Structures in the Anatomy of the Nose, Throat and Ear

A three period course — $4.50

Limited to 35

First Lecture:

Surgical anatomy of the nose; the relationships of the turbinates, the meatuses and the ostia; the paranasal sinuses; the structure of the nasal septum; the blood supply and innervation of the mucous membrane of the nasal cavity; the relationship of the opening of the eustachian tube to surrounding structures; the lymphatic drainage of the nose.

Second Lecture:

Surgical anatomy of the pharynx; relationships and blood supply of the palatine tonsil and the pillars of the fauces; the relations of the muscles of the soft palate; the structure of the the nasal pharynx with a consideration of lymphoid tissue, especial attention being paid to the attachments of the superior constrictor of the pharynx.

Third Lecture:

The anatomy of the middle ear; the structure of the tympanic membrane; the ossicles; the opening of the eustachian tube into the middle ear and the attic; consideration of the relationships of structures in the medial wall of the tympanic cavity; the anatomy of structures considered most important in operations upon the middle ear. Each lecture will consist of a description with charts and lantern slides for thirty-five to forty minutes, to be followed by a fifteen minute reel of kodachrome moving pictures. Dissections and models will be exhibited.

COURSE 54

Room 711 Periods M-2, T-2, W-2

WALTER HUGHSON, M.D.

Abington, Pa.

Audiometric Technics and the Fitting of Hearing Aids

A three period course — $4.50

Limited to 35

This course will involve a consideration of the methods of testing hearing and making accurate diagnoses by means of modern electric audiometric equipment. It is felt that all functional tests of hearing can be carried out with much greater accuracy by such methods than by the conventional and extremely inaccurate tuning fork, watch tick and spoken voice method.

The theory and practice of audiometry will be discussed in detail. Special tests such as "loudness balance" and "speech reception thresholds" will be described. The specific application of auditory masking will be explained. Group audiometry, modern methods of exposing malingering and finally the interpretation of audiograms will all be considered.

The tone field method of testing hearing in little children will be discussed. The method consists of the use of pure tones or a complex noise as the sound signal. Its use is particularly important in the examination of multiple disability cases such as deaf spastics.

The fitting of hearing aids is an exact science. The entire field of hearing aid characteristics as related to specific hearing loss will be analyzed. Psychologic and occupational factors will be evaluated and what a properly fitted hearing aid must be expected to do for the individual with impaired hearing. The performance of specific aids both in relation to air and bone conduction and make will be dealt with on the basis of patient approval.

COURSE 55

Room 734 **Periods M-2, T-2**

MARVIN F. JONES, M.D.
New York, N. Y.

The Causes of Dizziness
A two period course — $3.00

Limited to 35

Thirty-five causes of dizziness will be discussed. There are, of course, other causes which may be added but the ones named are definite. The conference will be conducted as a basic science plus a clinical hour. The first day will be spent in an exposition of the mechanisms involved together with the pathology and physiology of the equilibratory apparatus. The second period will be utilized discussing the establishment of a diagnosis in those patients whose symptoms include dizziness. The recognized and theoretical treatments used today will be given in detail. Some unpublished results of this work will also be presented.

COURSE 56

Room 736 **Periods M 2 & 3**

PHILIP E. MELTZER, M.D.
Boston, Mass.

Comparison of the Endaural and External Approach in Mastoid Surgery
A two period course — $3.00

Limited to 40

COURSE 57

Room 736 **Periods T 2 & 3**

PHILIP E. MELTZER, M.D.
Boston, Mass.

Comparison of the Endaural and External Approach in Mastoid Surgery
A two period course — $3.00

Limited to 40

COURSE 58

Room 736 **Periods W 2 & 3**

PHILIP E. MELTZER, M.D.
Boston, Mass.

Comparison of the Endaural and External Approach in Mastoid Surgery
A two period course — $3.00

Limited to 40

Dr. Meltzer's two hour course, "Comparison of the Endaural and External Approach in Mastoid Surgery," is being presented three times as Course 56, Course 57, and Course 58. **Select course number according to the day on which you wish to attend.**

This course will consist of a demonstration of the endaural approach to the regions of the temporal bone which may be involved in ear disease. There will be a discussion of the postauricular approach as compared with the endauricular. Emphasis will be laid on the technic of incision and instrumentation. Illustrated lecture, discussion and actual demonstration on cadavers will compose the procedure. Dr. Julius Lempert's models will be on view.

COURSE 59

Room 752 **Periods M 2 & 3**

WERNER MUELLER, M.D.
Boston, Mass.

The Diagnosis of Deafness
A two period course — $3.00

Limited to 40

COURSE 60

Room 752 **Periods T 2 & 3**

WERNER MUELLER, M.D.
Boston, Mass.

The Diagnosis of Deafness
A two period course — $3.00

Limited to 40

COURSE 61

Room 752 **Periods W 2 & 3**

WERNER MUELLER, M.D.
Boston, Mass.

The Diagnosis of Deafness
A two period course — $3.00

Limited to 40

Dr. Mueller's two hour course, "The Diagnosis of Deafness," is being presented three times as Course 59, Course 60 and Course 61. **Select course number according to the day on which you wish to attend.**

The most important phase of the problem of deafness is its recognition. In this course the diagnosis of deafness will be dealt with under the following headings:

1. History taking
2. The physical examination
3. Audiometry
4. Differential diagnosis

The subject will be presented from the standpoint of the practicing otologist. Special emphasis is laid on idiopathic deafness.

COURSE 62

Room 754 Periods M 1 & 2

SCOTT N. REGER, Ph.D.
Iowa City, Iowa

BY INVITATION

Audiometry and Hearing Aids
A two period course — $3.00

Limited to 40

COURSE 63

Room 754 Periods T 1 & 2

SCOTT N. REGER, Ph.D.
Iowa City, Iowa

BY INVITATION

Audiometry and Hearing Aids
A two period course — $3.00

Limited to 40

Dr. Reger's two hour course "Audiometry and Hearing Aids" is being presented twice as Course 62 and Course 63. **Select course number according to the day on which you wish to attend.**

I. Audiometry
   A. Technic of presentation of stimulus and recording of results.
   B. Bone conduction acuity measurement.
   C. Factors influencing accuracy of measurements: masking and noise.
   D. Interpretation of results.
   E. Malingering tests.

II. Hearing Aids
   A. Types of instruments.
   B. The audiogram in relation to the fitting of hearing aids; the minimum and maximum losses of hearing at which hearing aids cease to be effective.
   C. Controlled intensity, speech intelligibility and articulation test.
   D. Psychological factors influencing patient's reaction to hearing aids.

COURSE 64

Room 714 Periods T 1 & 2, W 1 & 2

S. E. ROBERTS, M.D.
Kansas City, Mo.

Biochemistry and Nutrition Applied to Daily Office Practice of Otolaryngology
A four period course — $6.00

Limited to 25

The course will be divided into two parts. Each part will emphasize the office treatment of various otolaryngological complaints.

The first part will be devoted to the nonoperative management of sinus disease. The second part will be other troubles in our specialty with emphasis placed on auditory neuritis. Nutritional and biochemical phases will be discussed in both.

COURSE 65

Room 710 Periods M-2, T-2, W-2

GEORGE E. SHAMBAUGH, JR., M.D.
Chicago, Ill.

Fenestration Operation for Otosclerosis
A three period course — $4.50

Limited to 40

First Period:

Development and structure of the labyrinthine capsule. Histology of otosclerosis. Clinical diagnosis of otosclerosis. Theoretical considerations underlying the fenestration operation correlated with results of experimental fenestration on the monkey. Indications for fenestration operation.

Second Period:

Technic of the fenestration operation

a. Preoperative preparation of the patient.
b. Anesthesia.
c. Preparation of operative cavity to gain access to the labyrinth.
d. Preparation of plastic skin flap.
e. Creation of the labyrinthine fistula.
f. Control of infection and hemorrhage.
g. After care.

Third Period:

Results of the fenestration operation. Detailed analysis and study of results in more than 600 consecutive fenestration operations, and 30 revisions with a special reference to:

a. Operative and postoperative complications.
b. Cases where hearing was made worse.
c. Cases where hearing improvement was lost due to closure of the fenestra.
d. Effect of the operation on tinnitus.
e. Effect of the operation on the progression of the hearing defect.
f. Comparison of the improvements obtained with the hearing from a hearing aid.
g. Effect on the hearing of:
   1) Intercurrent secretory otitis media.
   2) Intercurrent suppurative otitis media.
   3) Pregnancy.

COURSE 66

Room 781 **Periods M-3, T-3**

S. R. SKILLERN, JR., M.D.
Philadelphia, Pa.

Treatment of Acute and Chronic Frontal Sinusitis with Its Complications

A two period course — $3.00

Limited to 30

Review of frontal bone relative to benign or malignant osteomyelitis of sinus origin. Etiology of sinus infections and their relation to intracranial and intraosseous tissue. Treatment for acute and chronic sinus infections by conservative or radical surgery. Surgery for osteomyelitis. 16 mm. movie.

COURSE 67

Room 716 **Periods M 1 & 2**

HORACE J. WILLIAMS, M.D.
Philadelphia, Pa.

Acute Suppurative Otitis Media and Chronic Mastoiditis

A two period course — $3.00

Limited to 30

COURSE 68

Room 781 **Periods W 1 & 2**

HORACE J. WILLIAMS, M.D.
Philadelphia, Pa.

Acute Suppurative Otitis Media and Chronic Mastoiditis

A two period course — $3.00

Limited to 30

Dr. Williams' two hour course, "Acute Suppurative Otitis Media and Chronic Mastoiditis," is being presented twice as Course 67 and Course 68. **Select course number according to the day on which you wish to attend.**

Acute Suppurative Otitis Media:

1. Embryological development of the middle ear.
2. The etiology of the infection which reaches the middle ear.
3. The changes in the virulence of various organisms during certain years.
4. The change in the mucosa of the middle ear during infection.
5. The use of sulfa drugs.
6. The use of penicillin in acute suppurative otitis media with early signs of meningitis.

Chronic Mastoiditis

1. Embryological development of the mastoid.
2. The etiology of the various types of mastoid.
3. The location of the perforation which causes chronic suppurative mastoiditis.
4. The pathology in the middle ear and mastoid.
5. Treatment, local and surgical.

Individual Courses

OPHTHALMOLOGY

1944

COURSE 201

Room 710 **Periods M-3; T-3; W-3**

A. W. ADSON, M.D.
Rochester, Minn.

BY INVITATION

The Differential Diagnosis and Treatment of Neuralgias of the Face

The results accomplished in the treatment of facial pains is in direct proportion to the accuracy of the diagnosis and the appropriate treatment instituted. The two major neuralgias are trigeminal and glossopharyngeal involving the fifth and ninth cranial nerves respectively. The attacks of pain are similar though the areas

involved differ. Both neuralgias are cured by intracranial section of the nerve affected. Alcohol injected into the peripheral branches of the fifth nerve results in temporary relief. This procedure is not suited for glossopharyngeal neuralgia because of the close proximity of the vagus nerve. The treatment of other facial pain is designed to relieve the particular cause.

COURSE 202

Room 739 **Periods M-1; T-1**

BENNETT Y. ALVIS, M.D.
St. Louis, Mo.

Acute Corneal Lesions — Diagnosis and Treatment

1. Therapeutic agents: Anesthetics, antiseptics, stimulants, cauterants. Surgery.
2. Injuries: Foreign bodies, abrasions, lacerated and incised wounds, infections, burns (thermal, chemical, radiation), exposure keratitis.
3. Inflammations:
   Ulcers — simple, hypopyon, ring, Mooren's.
   Virus diseases — herpes, simplex and zoster; dendritic ulcers; superficial punctate keratitis.
   Keratitis filamentosa, phlyctenular disease, avitaminosis, interstitial keratitis.
4. Fever therapy.

COURSE 203

Room 714 **Periods T-3; W-3**

CHARLES A. BAHN, M.D.
New Orleans, La.

Fundamentals of Ophthalmic Diagnosis

First, the technic of the ophthalmic examination should be simple and elastic but complete. Thus the normal will be accurately separated from the positively abnormal or doubtful. The information thus gained is then translated into terms of basic pathology, forming the rough pattern of diagnosis. Causation is then added to complete the pattern, which in essence is the accurate diagnosis. This plan is adapted to the simplest as well as most complex diagnostic problems, as will be demonstrated.

COURSE 204

Room 744 **Periods M-2; T-2**

S. JUDD BEACH, M.D.
Portland, Maine

Refinements of Refraction

Low myopia, usually regarded as of no particular significance, is emerging as one of the problem children of ophthalmology. Entirely apart from the fallacy that 20/20 vision is impossible in this condition, Selective Service examinations have shown a curious instability of central visual acuity. Investigation reveals a characteristic symptomatology, usually overlooked because of an assumed immunity to eye-strain in low myopes. Presbyopia also presents complications peculiar to this condition. Diagnosis and treatment will be discussed.

COURSE 205

Room 778 **Periods M-1; T-1; W-1**

PAUL BOEDER, Ph.D.
Southbridge, Mass.
BY INVITATION

Aniseikonia

A clarification of the concept of aniseikonia will be followed by a description of the means of measurement and correction. Some statistics will be presented. The effect of aniseikonia on binocular space perception will be discussed in detail, and the possibility of a functional test of aniseikonia will be outlined.

COURSE 206

Room 751 **Periods M-1; W-1**

ALBERT L. BROWN, M.D.
Cincinnati, Ohio

Ocular Tuberculosis

Clinical and pathological findings initiated the present concept of ocular tuberculosis as a local manifestation of a systemic disease. It usually occurs in apparently healthy individuals with healed or benign infection, usually with glands in the chest which are capable either of disseminating infection or sensitizing the individual.

Clinical manifestations and treatment will be discussed.

COURSE 207

Room 739 **Periods M-2; T-2**

FRANK E. BURCH, M.D.
St. Paul, Minn.

End Results in Retinal Detachment

What types of cases are most favorable for successful operation for retinal detachment?

What is a good result and what must one consider a failure in reporting results?

The compartive results in patients with myopia, high myopia, previous fundus pathology, post-trauma, aphakia and other complications will be reviewed. Also, the results incident to age of patient at time detachment occurs, extent of detachment, various types and location of tears or holes, or disinsertions, will be discussed.

The type of operation done, variations in technic and reasons for redetachment are considerations which affect the "end results."

COURSE 208

Room 732 **Periods T-2; W-2**

GRADY CLAY, M.D.
Atlanta, Georgia

Ophthalmoscopic Classification of Various Hypertensive Diseases

The subject will be illustrated with photographs of the various types of lesions. Angiospasm, arteriolarsclerosis and hypertensive retinopathies will be discussed with a presentation of a workable classification for various hypertensive diseases.

COURSE 209

Room 718 **Periods M-2; T-2**

SYBIL COX, A.M.
Cleveland, Ohio

BY INVITATION

The Causes, Effects and Remedying of Reading Difficulties

The lecturer will discuss the most common causes of reading disability including eye difficulties; the unfortunate effects of non-reading on the child's personality and behavior; and the recent trends in remedial instruction.

COURSE 210

Room 716 **Periods T-1; W-1**

WILLIAM H. CRISP, M.D.
Denver, Colo.

Cross Cylinders

1. Principles and technic of cross cylinders for astigmatic axis and strength.
2. Sources of error in testing for strength of cylinder.
3. Coordination of astigmatic tests with use of fogging method.
4. Use of strong cross cylinders for amblyopic eyes.
5. Relation between cross cylinders and astigmatic dials as to the cylinder axis and strength.

COURSE 211

Room 725 **Periods T-2; W-3**

RAY K. DAILY, M.D.
Houston, Texas

Physical Therapy in Ophthalmology

The physiologic action of physical therapy agents will be briefly reviewed. Indications for their use in surgical and medical ophthalmology will be outlined. The basis for detailed exposition will be formed by illustrations of the technic and practical application of these agents in daily office practice.

COURSE 212

Room 702 **Periods T-2; W-2**

WINDSOR S. DAVIES, Lt., M.C.
San Diego, Calif.

Optic Discs — Comparison of Clinical and Pathological Changes

This course will consist of the study of various congenital and acquired defects of the optic nerve head. The pathological changes occurring in these defects will be discussed and a correlation made between the clinical and pathological findings. Illustration will be by lantern slide.

COURSE 213

Room 713 **Periods M-1; T-1**

ELLIS FREEMAN, Sc.D.
Southbridge, Mass.

BY INVITATION

Recent Psychological Advances Bearing on Ophthalmology

Synergy is well understood in medicine. The physical sciences have been developing in a similar direction, represented by the emphasis on relationships as against "atomoid" units. Gestalt psychology also has been especially productive in experimental data during the past twenty years, and could enrich the concept of synergy in ophthalmology and have a bearing on practice. Attention to these data would, accordingly, help keep ophthalmology in step with developments that are of profound influence on the basic principles of all sciences and technologies.

COURSE 214

Room 751 Periods M-3; T-3

RICHARD C. GAMBLE, M.D.
Chicago, Ill.

Acute Inflammation of the Orbit

The subject of orbital inflammation will be discussed with special emphasis on the way a thorough understanding of the etiology and pathologic anatomy influence the choice of treatment for this condition.

COURSE 215

Room 751 Periods M-2; W-2

J. CONRAD GEMEROY, M.D.
Detroit, Mich.

External Diseases of the Eye

This lecture on external eye diseases is illustrated in color and shows usual and some unusual diseases of the lids, conjunctiva, and cornea with discussion of their diagnoses and treatments. The lecture will be illustrated throughout with kodachrome slides.

COURSE 216

Room 732 Periods M-1; T-1

T. R. GITTINS, M.D.
Sioux City, Iowa

Headaches as an Eye, Ear, Nose and Throat Problem

Classification and differential diagnosis are of great importance if treatment is to be rational and reasonably successful.

Periodic headaches of vasomotor origin with intervals of good health are the most common of all types and continually are confused in diagnosis and treatment in the every day office practice of ophthalmology and otolaryngology.

COURSE 217

Room 751 Periods T-1; W-3

GEORGE GUIBOR, M.D.
Chicago, Ill.

The Value of Orthoptics in Private Practice

The treatment of any oculomotor disturbance must rest upon an accurate diagnosis. A mere symptomatic diagnosis, "esotropia" or "esophoria" for example, is not sufficient for adequate treatment because the underlying cause of the oculomotor defect has not been ascertained. The examiner, therefore, must recognize deviations as a symptom of neuromotor derangement rather than a disturbance in fusion or a defect of a single muscle.

Many deviations ordinarily considered comitant are really the result of injury to supranuclear pathways or are derangements of synkinetic neuromotor connections. These are amenable to nonsurgical treatment if it is undertaken before contractures of the extraocular muscles occur. A discussion of supranuclear deviations in children and their diagnosis and treatment will be undertaken.

The use of prisms prescribed from a neurologic viewpoint will be demonstrated.

COURSE 218

Room 713 Periods M-3; T-3

PARKER HEATH, M.D.
Detroit, Mich.

Chemotherapy

This course will be a discussion of chemotherapy in general and as a treatment in certain diseases particularly common in ophthalmology. Various chemical and antibiotic agents will be commented upon. Some possible points of action will be outlined. Methods of administration will be considered. Certain hazards and precautions, necessary and important, will be stressed. The treatment of some ocular diseases will be described.

COURSE 219

Room 756 Periods M-1; T-1

BAYARD T. HORTON, M.D.
Rochester, Minn.

BY INVITATION

Headaches and Head Pains Clinical Varieties and Therapeutic Suggestions

A general discussion of the differential diagnosis of the various types of headaches and atypical head pains, accompanied by a technicolored motion picture demonstration of the clinical features, location and distribution, as well as associated ophthalmologic and rhinologic manifestations.

COURSE 220

Room 718 Periods M-1; T-1

HAROLD H. JOY, M.D.
Syracuse, N. Y.

Chronic Uveitis Including Sympathetic Ophthalmia

The various types of chronic uveitis will be considered with special emphasis on tuberculous lesions and sympathetic ophthalmia. The etiology, pathology, clinical course and prognosis will be discussed. Chief consideration will be given to the differential diagnosis and treatment of the disease, and its complications and sequelae.

COURSE 221

Room 739 Periods M-3; T-3

PETER C. KRONFELD, M.D.
Chicago, Ill.

Gonioscopy

The present status of gonioscopy may be summed up by listing some of its plus as well as its minus values.

Plus Values of Gonioscopy:

1. Gonioscopy offers a simple mechanical explanation for certain glaucomas.
2. Gonioscopy helps in choosing the type of operation most suitable for the case under consideration.
3. Gonioscopy helps in recognizing the causes of failure of glaucoma operations.
4. The information obtained by gonioscopy may be of value in inflammatory, traumatic and neoplastic diseases of the eye.

Minus Values of Gonioscopy:

1 Gonioscopy offers no explanation of the mechanism of certain glaucomas.
2. Gonioscopy requires some special equipment. From two to four hours are required to learn the technic of gonioscopy. Considerable experience is required to interpret cases in which the landmarks have been obliterated.
3. Gonioscopy may be conducive to a purely mechanical concept of glaucoma.

COURSE 222

Room 702 Periods M-1; T-1

HEDWIG S. KUHN, M.D.
Hammond, Ind.

Visual Job Analysis
Refraction and Correction for the Job

A careful job analysis is basic to the understanding of correcting refractive errors for people working in industry, where they are confronted with an infinite variety of jobs. Procedures in evaluating the needs of an industrial eye program include such items as inspection of the operator of a machine, checking his work distances, observing whether or not his work is in motion and if stereopsis is essential for safety and efficiency. The refraction is then made on the basis of such information. Illustrations and discussion of different types of industrial operations will be followed by the suggested practical additions to refractive technics to be studied, also the corrections of whatever visual defects occur which interfere with the operation of any or all machines. The following are some of the elements basic in consideration of the technic for refraction and in determining the final prescription: whether or not a person must climb, why correcting a myope for an 8 inch job is totally different from the correction of a crane operator working at 150 feet; complications of corrective refractive errors in certain jobs; problems associated with bifocals; the correction of refractive errors in people over forty, necessary near-point vision data to be obtained; the relationship of proper correction to production and rejects of material; also a fully illustrated discussion of problems closely connected with salvaging of applicants with visual defects as basis for pre-employment placement.

COURSE 223

Room 739 Periods T-1; W-2

ALBERT N. LEMOINE, M.D.
Kansas City, Mo.

Allergy in Ophthalmology

The lecturer will discuss the following:

Some underlying causes of allergy, including biochemical and physiological aspects.

Diagnosis: History, tests with allergens and by elimination of allergens.

Treatment: Local and general medical treatment.

COURSE 224

Room 721 Periods M-3; W-3

RALPH I. LLOYD, M.D.
Brooklyn, N. Y.

Atrophy of the Tapetum with Sclerosis of the Choroidal Vessels

A discussion of atrophy of the pigment layer of the retina and the choriocapillaris with sclerosis of the vessels of the choroid. The milder cases seen daily show but little loss of vision; in advanced cases with marked changes in the choroidal vessels, central vision often persists under seemingly impossible conditions.

COURSE 225

Room 702 Periods M-3; T-3; W-3

AUREL E. MANGOLD, Capt., M.C.
New York, N. Y.

BY INVITATION

Types and Preferences in Lens Forms and Fitting of Spectacles

This discussion will stress the necessity of proper selection of ophthalmic lenses, explaining how and why an improper lens form may impair the results of an expert refraction.

It will deal with the importance of interpupillary distances, near and far, and compensation for vertical imbalances in anisometropia. It will also include a discussion of the effect of lens forms as related to aniseikonia, cataract, and high dioptric prescriptions. The proper selection of types and sizes of bifocals will be dealt with as well as colored lenses and their values.

The method of taking correct facial measurements will be stressed, i.e., interpupillary distances, size of lens, bridge measurements, and length of temple. Errors in adjustment of the finished glasses will be discussed and the corrections explained.

COURSE 226

Room 720 Periods M-1; T-1; W-1

PAUL G. MOORE, M.D.
Cleveland, Ohio

The Management of Intraocular Foreign Body Cases

A discussion of foreign bodies, the diagnosis, and methods used in their removal.

1. Importance of history and record.
2. Diagnosis.
3. Roentgen ray examination and technic.
4. Hospitalization.
5. Operative procedure.
   (a) Anterior route.
   (b) Posterior route.
   (c) Type of incision.
6. Type of magnet; giant or hand magnet.
7. Liability of detachment of retina and its prevention.
8. Length of hospitalization.
9. Length of disability.
10. Percentage of and indications for enucleations.
11. Results in visual acuity.

COURSE 227

Room 720 Periods T-3; W-3

W. HOWARD MORRISON, M.D.
Omaha, Neb.

Malingering

Quick, reliable and easily performed tests for malingering are very important, particularly today. The increasing number of persons employed in hazardous industries and war work carries with it a proportionate increase in injuries and simulated injuries to the visual apparatus. An evaluation of the numerous tests for malingering will be made, and those tests especially suitable for present day practice will be demonstrated.

COURSE 228

Room 740 Periods M-1; W-1

R. TOWNLEY PATON, M.D.
New York, N. Y.

Keratoplasty

A practical demonstration will be given of the corneal grafting operation illustrating the various commonly employed procedures. Considerable attention will be given to the indications, contraindications, and expected results in carefully selected cases. Lantern slides and moving pictures will be used to clarify these points.

COURSE 229

Room 740 **Periods T-1; W-2**

DOHRMANN K. PISCHEL, M.D.
San Francisco, Calif.

Retinal Detachment
Preoperative, Operative, and Postoperative Care

The mechanism of the development of a retinal detachment will be discussed from a practical standpoint, so that the steps necessary in its treatment will be understood. Methods of examination necessary for the discovery of tears or holes and their localization will be considered. Important points in the preoperative care will be taken up. The diathermy operation for retinal detachments will be discussed in some detail, with especial attention to practical points. In conclusion, postoperative care and the duration of such care will be considered.

COURSE 230

Room 721 **Periods T-2; W-2**

BENJAMIN RONES, M.D.
Washington, D. C.

Corneal Dystrophies

Corneal dystrophies will be differentiated according to their developmental and clinical patterns. The various etiological theories will be discussed. Recent investigations on corneal physiology will be integrated with possible etiology. Therapy, although primitive, offers suggestive possibilities.

COURSE 231

Room 725 **Periods M-3; T-3**

C. W. RUTHERFORD, M.D.
Indianapolis, Ind.

Myopia

1. Myopia as a refractive state: age at onset; period of progress; age of stabilization; treatment.

2. Myopia as a disease or complication: etiology; associated conditions such as endocrine imbalance or insufficiency, overweight and overgrowth, faulty hygiene and diet, injurious postures, pernicious visual habits; signs and symptoms; anatomic and pathologic changes; treatment.

COURSE 232

Room 725 **Periods M-2; W-2**

SCOTT STERLING, A.B.
Rochester, N. Y.

BY INVITATION

Application of Spectacle Lenses
Their Development and Use
Theory of Bifocals

A pair of glasses must accomplish several things if it is to give the wearer clear, effortless vision over protracted periods of time and permit him to carry on his work and his play with the maximum amount of comfort and satisfaction.

The prescriptions written for soldiers show an amazingly large number of strong powers. Also, the necessary correction of myopia among soldiers shows that the same amount of myopia among civilians has been all too frequently neglected. The use of colored glasses, selective glass filters and their application in military and civil life will be discussed. The application of bifocal lenses for anisometropic corrections and various special purpose bifocal and trifocal lenses will be discussed.

COURSE 233

Room 713 **Periods T-2; W-2**

GEORGE H. STINE, M.D.
Colorado Springs, Colo.

Differential Diagnosis and Intraocular Localization of Retinal Detachment

Preoperative management of retinal detachment cases, including differential diagnosis and localization of retinal tears, will be discussed in detail. Simple but precise methods of localization will be described, and helpful, practical points outlined. Lantern slides will be shown, and illustrative cases used.

COURSE 234

Room 756 **Periods M-2; T-2**

ARNO E. TOWN, Lt. Comdr., M.C., U.S.N.R.
Bethesda, Md.

Contact Lenses

Contact lenses are being recognized more and more as valuable therapeutic agents in keratoconus, corneal dystrophise, keratitis and lid conditions. This course covers the details of the contact glass refraction, the taking of the impressions and the making of the positive from which the glass is constructed.

COURSE 235

Room 751 **Period T-2**

ROBERT VON DER HEYDT, M.D.
Chicago, Ill.

Advanced Slit Lamp Microscopy

The following conditions will be discussed and shown with colored photographs and illustrations:

1. Corneal stromal opacification.
2. Arcus senilis.
3. Arcus senilis — stromal lines in.
4. Sulcus marginalis senilis.
5. Limbus girdle (pathological?)
6. Lineae cornea senilis. (Staehli line.)
7. Hassal-Henle Descemiti warts (peripheral).
8. Cornea guttata (central).
9. Cornea farinata.
10. Limbus pigmentation and vessel sclerosis.
11. Corneal pigmentation at limbus.
12. Depigmentation on posterior cornea.
13. Depigmentation of pupillary collar of iris.
14. New pigment on iris.
15. Capsular exfoliation.
16. Anterior suture punctation.
17. Lens sclerosis (double focus).
18. Vitreous changes.

COURSE 236

Room 720 **Periods M-3; W-2**

HAROLD F. WHALMAN, M.D.
Los Angeles, Calif.

Calibration and Application of the Shahan Thermophore in Ophthalmic Conditions

The Shahan thermophore affords a valuable means of exactly controlled heat application in ophthalmic conditions. The instrument requires calibration, however, to obtain this exactness, as the degree of heat delivered at the applicator tip varies with the size of the tip. A method of calibration will be demonstrated, and the heat necessary in treatment of a variety of conditions will be outlined in accordance with the lecturer's own experience.

Individual Courses

OTOLARYNGOLOGY
1944

COURSE 301

Room 731 **Periods M-1; T-1; W-1**

OSCAR V. BATSON, M.D.
Philadelphia, Pa.
Anatomic Review of Nose

COURSE 302

Room 731 **Periods M-2; T-2; W-2**

OSCAR V. BATSON, M.D.
Anatomic Review of Pharynx and Larynx

COURSE 303

Room 731 **Periods M-3; T-3; W-3**

OSCAR V. BATSON, M.D.
Anatomic Review of the Ear

The practical anatomy of the several regions indicated, with particular reference to diagnosis, instrumentation, and surgical intervention. The illustrative material will be from anatomic specimens or from color slides of anatomic specimens. In each one of the subdivisions, the spread of infections from the particular area into adjacent regions will be discussed as well as the invasion of these regions from without. In **Course 301** the commoner variation of the paranasal sinuses will be shown as well as the variations in the nasal fossae and the anatomy of local anesthesia. In **Course 302** there will be a discussion of the soft palate, and a classification of the tracheobronchial tree. In **Course 303** the essential anatomic features of the several classes of ear operations will be presented as well as the anatomy of the temporal bone and its radiologic identification.

COURSE 304

Room 778 **Periods M-2; T-2; W-2**

AUGUST L. BECK, M.D.
New Rochelle, N. Y.

Management of Common Disorders Encountered in Office and Hospital Practice

Epistaxis

1. Source in nose.
2. Immediate management.
3. Follow-up.

Technic of administering local anesthesia for intranasal and sinus operations.

Middle turbinectomy technic. Instrument for same.

Treatment of syncope during intranasal operation under local anesthesia.

Motion picture demonstration of antrum irrigation technic.

Removal of calculus from salivary ducts, with illustration by lantern slides.

Differential diagnosis of tumefactions of the salivary glands.

1. Nonsuppurative inflammatory tumefactions.
2. Suppurative tumefactions.
3. New growths.

Neck infections including motion picture demonstration of approaches.

Motion picture demonstration of fistula test.

COURSE 305

Room 724 Periods M-2; T-2; W-2

PAUL A. CAMPBELL, Lt. Col., M.C.
Randolph Field, Texas

Aviation Otolaryngology

More than 80 per cent of the complaints and discomfort associated with flying have otolaryngological significance. The ear is most vulnerable due to the effects of noise both audible and inaudible, positive and negative pressure changes due to altitudinal variation, and the effects of lessened oxygen supply to the organ of Corti. The sinuses are affected, sometimes to severe degrees if their ventilation is abnormal. The effects of positive and negative pressure changes on mucous membrane lined cavities will be demonstrated. Effect of parachute jumping and power diving will be discussed. Otolaryngological qualifications for the U. S. Army Air Forces will be considered.

COURSE 306

Room 705 Periods M-1; T-1; W-1

THOMAS E. CARMODY, M.D.
Denver, Colo.

Oral Cavity in Health and Disease

Examination of lips and mouth.

Complete examination of teeth and gums. This should also include roentgenograms of teeth and jaws.

Roentgenograms of maxillary sinuses should be studied in connection with those of teeth as infection of these sinuses may be transmitted from or to the teeth and alveolus.

Examination of tongue and floor of mouth including the ducts of the salivary glands.

Palpation both external and internal for enlarged glands and lymph nodes.

Roentgen ray of salivary glands for calculus.

The character of the mucous membrane as to color consistancy and secretion.

Careful examination of attachments of tongue to floor of mouth and to posterior structures, even the base, and note made of entrance to pharynx and larynx. This cannot be done without a good light and a laryngeal or mouth mirror.

The palate with its attachments should also be carefully noted.

COURSE 307

Room 778 Periods M-3; T-3; W-3

FRED W. DIXON, M.D.
Cleveland, Ohio

Anatomy and Treatment of Acute and Chronic Sinusitis

It is essential in the conservative or operative treatment of the nasal accessory sinuses to have a thorough knowledge of their anatomy.

For those who do not find the dissecting room available, this course is presented. One hundred specimens showing the various abnormalities of the sinuses will be examined; the remaining time will be spent in discussing treatment and operations.

COURSE 308

Room 776 Periods M-1; T-1; W-1

J. WINSTON FOWLKES, M.D.
New York, N. Y.

Diagnosis and Treatment of Mastoiditis

1. General symptoms of ear disease.
2. Signs and symptoms of mastoiditis.
3. Pathology of acute and chronic mastoiditis.
4. Bacteriology.
5. Roentgen ray findings.
6. Mastoiditis in children as compared with adults.
7. Sulfa therapy.
8. Intracranial complications.
9. Types of operations.
10. Postoperative care.

COURSE 309

Room 724 Periods M-1; T-1; W-1

A. C. FURSTENBERG, M.D.
Ann Arbor, Mich.

Reconstruction of the Facial Nerve

A plea is made for the preservation of the facial nerve in operations upon the parotid gland. One need not despair of maintaining its fibers intact even when large infiltrating lesions of the gland are encountered.

An optimistic point of view in regard to safeguarding the facial nerve can best be maintained by an initial exposure near its emergence from the stylomastoid foramen. Thus early identification of the nerve is made possible and its important ramifications traced with facility throughout the parotid gland.

The advantage of an end to end anastomosis over nerve transplant seems obvious. The tortuous route of the facial nerve can be appreciably shortened by lifting it out of its bony canal and freeing it along its course in the parotid gland. The union of the ends of a severed nerve which at first seems futile may often be accomplished by this method.

COURSE 310

Room 740 Periods M-2; T-2

T. C. GALLOWAY, M.D.
Evanston, Ill.

Laryngeal Emergencies Including Bulbar Poliomyelitis

This course will include indications, technic and timing of intubation and tracheotomy; pathology associated with obstructions including tracheo-bronchitis; and after-care including postural drainage, irrigation and endoscopy for aspiration and plug removal. Flame injuries, respiratory tract and their treatment.

COURSE 311

Room 720 Periods M-2; T-2

E. T. GATEWOOD, M.D.
Richmond, Va.

Roentgen Ray Interpretation of Sinus Disease

Why the rhinologist should make and interpret his patients' films. Pitfalls in the interpretation of sinus films. Clinical and roentgenographic correlation. Different types of sinus disease illustrated. Pathologic processes including new growth, osteomyelitis, hyperostosis frontalis interna presented. Evaluation of sinus films with and without iodized oil mapping as an aid in selecting surgical methods.

COURSE 312

Room 776 Periods M-3; T-3; W-3

WILLIAM D. GILL, M.D.
San Antonio, Texas

Fractures of the Facial Bones

A brief anatomic review of the anatomy of the facial bones followed by a discussion of the various causes producing fractures in this region. A classification of the various types of fractures encountered with emphasis on certain types and their relationship to the nasal accessory sinuses and orbital structures will be considered. The clinical course and complications of such injuries and new ideas which have been developed recently in the treatment of such fractures will be reviewed. The various methods of fixation of fragments will be considered as well as a variety of methods of reduction of fragments. Plastic procedures to correct loss of substance will be briefly discussed.

COURSE 313

Room 718 Periods T-3; W-3

W. E. GROVE, M.D.
Milwaukee, Wis.

Anesthesia in Otolaryngology

In operative procedures in ear, nose and throat, local anesthesia is more frequently used than general anesthesia. The course will discuss the choice of either local or general anesthesia, the local anesthetics in common use, the advantages and disadvantages of local anesthesia, the question of poisoning and its treatment, and the methods of applying local anesthesia by topical application, infiltration, and nerve blocking, together with a discussion of preliminary medication.

As the successful use of local anesthesia requires an accurate knowledge of sensory nerve supply, slides will be used to illustrate the sensory nerve supply of the various regions.

COURSE 314

Room 702 Periods M-2; W-1

J. SWIFT HANLEY, M.D.
New York, N. Y.

The Anatomy and Anomalies of the Temporal Bone

The object of this course is to demonstrate with lantern slides the normal anatomy and anatomic variations found in a large number of temporal bones.

The anatomy will be presented in its relation to pathologic conditions encountered and surgical procedures to be followed.

COURSE 315

Room 728 Periods M-1; T-1; W-1

FRENCH K. HANSEL, M.D.
St. Louis, Mo.

Allergy of the Nose and Paranasal Sinuses

In the discussion of this subject will be considered the fundamental principles of diagnosis and treatment. A new rapid technic of staining ocular, nasal and bronchial secretions will be demonstrated. Among the various methods of treatment, detailed consideration wil be given to dust therapy.

COURSE 316

Room 740 Periods M-3; T-3
Room 756 Period W-3

FREDERICK T. HILL, M.D.
Waterville, Maine

The Management of Acute and Chronic Conditions of the Maxillary Antrum

The maxillary antrum is of especial significance to the otolaryngologist. It is the most frequently involved of the paranasal sinuses and is the most amenable to treatment.

Intelligent therapy depends upon an understanding of the applied anatomy and the physiology. An attempt will be made to evaluate some of the newer methods of treatment, based upon scientific findings.

The course will take up the diagnosis and treatment of acute and chronic conditions, including infections, injuries, antro-alveolar fistulae and neoplasms. Various operative procedures will be considered.

COURSE 317

Room 710 Periods M-1; T-1; W-1

JULIUS LEMPERT, M.D.
New York, N. Y.

Lempert Fenestra Nov-Ovalis Operations For the Improvement of Hearing in Clinical Otosclerosis: Indications, Problems Encountered, End Results

Though the origin of and the cure for the pathologic lesion known as otosclerosis have both remained unknown, middle ear deafness resulting from this pathologic lesion can be cured by creating a new oval window in the surgical dome of the vestibule. Slides and dissections of technic will be shown. The diagnosis of clinical otosclerosis will be reviewed. The surgical principle upon which this surgery is based will be presented. The present-day indications and contraindications will be given. Objectives aimed for as a result of this surgery will be outlined, and the end results obtained summarized. Problems involved in this surgery will be discussed, and methods employed to conquer them suggested. Which ear to operate, when to operate, and why operate will be discussed.

COURSE 318

Room 725 Periods M-1; T-1; W-1

J. R. LINDSAY, M.D.
Chicago, Ill.

Meniere's Syndrome: Diagnosis and Treatment

The course has been designed to cover the routine examination necessary in cases of vertigo, the differential diagnosis and treatment. It includes:

1. Definition of the terms Meniere's Symptom Complex, Meniere's disease, and pseudo-Meniere's Syndrome as commonly used.
2. Routine examination and method of recording findings.
3. Significance of the findings in localizing the site of disease.
   Significance of postural vertigo.
4. Differential diagnosis in so-called pseudo-Meniere's and Meniere's syndrome.
5. Discussion of Meniere's disease, including clinical stages, auditory characteristics, pathology and treatment.

COURSE 319

Room 750 Periods M-2; T-2; W-2

ROMEO A. LUONGO, M.D.
Philadelphia, Pa.

Practical Anatomical Landmarks and Surgical Anatomy of the Fronto-Ethmosphenoidal Region

Surgical, topographical and pathological anatomy of the region involved in the external fronto-ethmosphenoidal operation will be discussed in full detail and illustrated with drawings, lantern slides, wet and dry anatomical specimens and moving pictures.

Special emphasis will be placed upon extranasal and intranasal practical landmarks and important anatomical relationships which will expediate the technic and be of great value in keeping the operator always well oriented and sure of his way throughout the entire operative procedure.

The dangerous and most vital regions will be mapped out with great care and proper warnings will be given. Technical details and hints, which will make this surgical procedure comparatively safe, will be amply discussed.

COURSE 320

Room 721 Periods M-1; T-1; W-1

J. W. McCALL, M.D., Cleveland, Ohio

Laryngectomy and Voice Training

Early cancer of the larynx has only one symptom, hoarseness. Pain and difficulty in breathing are later symptoms. The frequently applied label of chronic laryngitis to those cases of carcinoma of the larynx has been the cause of much suffering and many deaths.

When a patient around the age of 40 or older presents himself with a history of hoarseness of over two weeks' duration, suspect a carcinoma of the vocal cord until you prove otherwise.

DIAGNOSIS: Mirror laryngoscopy is easy and simple to use and should be done on every patient seen by a laryngologist. If mirror laryngoscopy reveals a growth in the larynx, this should be removed and a microscopic examination made. In doing a mirror laryngoscopy, it is important to look at the movements of the vocal cords. A limitation of motion of one cord is very suggestive of a malignancy.

In doing a biopsy great care should be used to obtain the tumor mass and a sufficiently deep bite to secure a complete microscopic examination.

In no other organ of the body does carcinoma yield so successfully to early diagnosis and surgical treatment as in the larynx.

The decision between laryngofissure and laryngectomy is not always easy, but if laryngeal surgeons would remember not to compromise with carcinoma, many more patients with cancer of the larynx would survive.

Fortunate is the surgeon who has one or more patients who have developed a good esophageal voice. It is of inestimable value to the patient contemplating laryngectomy to see and talk to someone who has had the operation and has developed an esophageal voice. Persons who have accomplished this inspire confidence, improve morale and stimulate hope in the one who is about to undergo this ordeal. A patient with an esophageal voice is far more successful in "selling" the operation than is the surgeon.

Slides showing fluoroscopic view of a laryngectomized patient during phonation.

COURSE 321

Room 744 Periods M-3; T-3

BERNARD J. McMAHON, M.D.
St. Louis, Mo.

Treatment of Sinusitis in Children

Sinusitis in children is a definite clinical entity of which we must take serious cognizance, if we are to save the child many years of partial disability and protect it against the more serious and intractible complications and sequelae which may be established in the middle ears, the lungs or the kidneys. The intelligent treatment of a sinusitis presupposes an adequate, if not a thorough, knowledge of the anatomy, the physiology, and the pathology of the nose and sinuses of the child. It is therefore my intention to review these subjects as a necessary preliminary step to a discussion of the disease process which is leading to a better understanding of the rationale of treatment, planned to readjust this relationship to its normal status.

COURSE 322

Room 740 Period M-1

HARRIS P. MOSHER, M.D.
Marblehead, Mass.

My Milestones

This is a reminiscent paper. The following topics will be discussed briefly, and often dogmatically.

The frontal sinus
Extradural abscess
Brain abscess.
Arachnoid cyst.
Orbital abscess.
The lachrymal sac operation.
Pus in the neck.
Fibrosis of the esophagus.
Dilation of the terminal portion of the esophagus by fluoroscopic illumination.
Cervical exostoses. The lateral roentgenogram of the neck.
Infectious thrombophlebitis.
Tracheotomy without a tube.

COURSE 323

Room 722 Periods M-3; T-3; W-3

C. STEWART NASH, M.D.
Rochester, N. Y.

The Functional Diseases of the Nose

It has been estimated that about 90 per cent of the diseases of the nose are physiologic in origin.

If the normal functions of the nose are to be maintained, the nasal mucosa must be normally active; it can neither hypofunction nor hyperfunction.

This course will include informal lectures and discussions on such hypofunctioning diseases as physical, psychic, and febrile rhinitis; and such hyperfunctioning diseases as irritative, infective (the common cold) and endocrine rhinitis. Emphasis is to be laid on conditions which are often unrecognized and consequently improperly treated.

COURSE 324

Room 750 Periods M-3; T-3

M. D. RYAN, M.D., New York, N. Y.

Diagnosis and Treatment of Laryngeal and Intrabronchial Tuberculosis

1. General considerations.
2. Signs and symptoms.
3. Pathology.
4. Bacteriology.
5. Roentgen ray findings with laminographic studies.
6. Indications and contraindications for thoracic surgery.
7. Types of operation.
8. Treatment and prognosis.

COURSE 325

Room 750 Periods M-1; T-1; W-1

SAMUEL SALINGER, M.D.
Chicago, Ill.

Deformities of the Nasal Septum

1. A brief summary of the embryology of the septum and how its component parts are developed.
2. Analysis of the "Sphenoid and Huxley Angles" as bearing on the philogenesis of the septum and as pointing to possible reasons for deviations from the midline.
3. Etiology of septum deviations; various theories considered. Question as to the relative importance of developmental factors in contrast to acquired factors such as trauma.
4. Types of septum deviation; ages at which they may be observed and analysis of the location of bends and crests.
5. Surgical correction of septum deformities.
   a. The classic Killian operation.
   b. Freer's modifications.
   c. Metzenbaum's technic.
   d. Variations in technic according to indication.
6. Technic.
   a. Preparation.
   b. Anesthesia.
   c. Instruments.
   d. Closure.
   e. Postoperative dressing.
   f. Postoperative care.
   g. Complications.
7. Septum correction combined with plastic procedures about the nose.
   a. Correction of bony twist.
   b. Removal of hump.
   c. Shortening of the nose.

Illustrations, photographs and models will be provided to clarify many of the points mentioned above.

The classic Killian submucous resection is inapplicable and unsatisfactory in a great many cases because it fails to take into consideration such abnormal-

ities as dislocated septal border, deviations causing external deformity and deviations associated with other nasal abnormalities which are displeasing. As has been pointed out by many observers it is not always necessary nor wise to remove a large amount of cartilage and bone to provide the patient with adequate breathing space. It is just as important to conserve these tissues as far as possible as it is to remove the obstruction. The technic, therefore, should be directed to the attainment of maximum breathing space through minimal sacrifice of supporting structures.

COURSE 326

Room 718 Periods M-3; W-1

J. D. SINGLETON, M.D.
Dallas, Texas

Operative Procedures on the Temporal Bone Based on Surgical Anatomy

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The course will consist of a brief review of the practical surgical anatomy of the temporal bone, and the technic of the simple, modified radical and radical mastoidectomy, postauricular approach.

The anatomy of the overlying soft parts including the sensory nerve supply will be discussed briefly, also the technic and indications for local anesthesia in mastoidectomy. Important external and internal landmarks will be discussed in some detail. The original route of extension, important relations and final termination of unusual cell groups, including those extending into the petrous pyramid, and the separation of cells by dense septa into more or less isolated groups will be described in considerable detail.

A complete description of the course and relations of the facial nerve in the middle ear and mastoid portions of the temporal bone, and the surgical technic necessary to avoid injury to the nerve in the various operations will be given and stressed.

In discussing the technic of the above mentioned operations, the more difficult phases will be emphasized. Numerous photographic and roentgenographic slides will be shown during the course of the lecture.

COURSE 327

Room 724 Periods M-3; T-3; W-3

MAURICE F. SNITMAN, M.D.
Chicago, Ill.

Surgical Anatomy of the Neck

The discussion will be limited to the prevertebral portion of the neck with emphasis on the surgical aspects. Sufficient cadaver material and slides will be used to make this course as eminently practical as possible.

The surgical aspects will be concerned chiefly with the following procedures:

a. The approach to the viscero-vertebral angle, which is necessary for ligations of large vessels of the neck, drainage of deep neck infections, bronchial cyst and pharyngeal diverticulum removal and block dissection of cervical lymphatics.
b. Laryngofissure
   Laryngectomy
   Tracheotomy

COURSE 328

Room 721 Periods M-2; T-3

LaVERNE B. SPAKE, M.D.
Kansas City, Kansas

Intranasal Surgical Anatomy

With slides, anatomical specimens, the blood and nerve supply operations.

COURSE 329

Room 732 Periods M-3; T-3; W-3

C. L. STRAITH, M.D.
Detroit, Mich.
BY INVITATION

W. B. SLAUGHTER, M.D.
Chicago, Ill.
BY INVITATION

Plastic Surgery of Facial Injuries

The treatment of all acutely injured persons must follow a rather definite course — the purpose of which is to give a maximum of benefit to the patient. To obtain this maximum benefit requires adherence to certain fundamental surgical principles, outlined as follows: (Illustrated with kodachrome slides and movies.)

1. Treatment of impending shock or actual shock.
2. Prevention of additional contamination and trauma to the wound. (Use of antiseptics.)

3. Estimation of the patient's general condition and recognition of the presence of multiple injuries.
4. Cleansing of the wound. (Prevention of "tattoo" marks, removal of foreign bodies, etc.)
5. Surgical care. In plastic surgery of the face certain problems are encountered peculiar to this region. These points will be stressed together with emphasis on fixation of fractures, suturing of wounds and reconstructive procedures for recent or old injuries.
6. After care.

COURSE 330

Room 713 Periods M-2; W-1

WALTER H. THEOBALD, M.D.
Chicago, Ill.

Infections of the Epipharynx Pathology, Etiology and Clinical Aspects with Special Reference to Treatment

The term, Thornwaldt's disease, is used in a broad sense to apply to all congenital and acquired fistulas, cysts and abscesses which occur in the epipharyngeal vault.

This area deserves routine examination and should be given careful study in cases of postnasal discharge, chronic or recurrent pharyngitis — persistent fevers, bleeding, cough, asthma, etc.

Direct examination of the epipharynx is simple and easily performed with the Yankhauer speculum. This will be demonstrated by movie films. The management and method of treatment both local and surgical will be discussed.

COURSE 331

Room 734 Period W-1

GABRIEL TUCKER, M.D.
Philadelphia, Pa.

Obstructive Dyspnea and Tracheotomy

Definition. For the purposes of this discussion, obstructive dyspnea may be defined as "Dyspnea due to mechanical obstruction of the airway through the pharynx, larynx and tracheobronchial tree."

Etiology. Obstruction of the airway may be caused by foreign body, inflammatory, neoplastic or traumatic lesions of the pharynx, larynx and tracheobronchial tree. The occurrence of obstructive dyspnea in the presence of such lesions creates an urgent indication for relief of obstruction and restoration of the airway. Where mechanical removal of obstruction by direct laryngoscopy or bronchoscopy will restore the airway, short-circuiting of the airway by tracheotomy may not be required. Where this is not practical, however, the indication for tracheotomy becomes urgent.

Tracheotomy. Tracheotomy is one of the oldest operative procedures known to surgery. In spite of this fact, the indications for tracheotomy in obstructive dyspnea are frequently unrecognized. The modern methods of temporary relief of obstructive dyspnea, and the prevention of mediastinal complications by the use of the bronchoscope or the Mosher Life-Saver during tracheotomy, are not fully appreciated. Tracheotomy, indications, technic and after-care will be demonstrated, with colored motion pictures and lantern slides.

COURSE 332

Room 776 Periods M-2; T-2; W-2

F. D. WOODWARD, M.D.
Charlottesville, Va.

The Management of Recent Faciomaxillary and Mandibular Fractures

The development of high speed transportation has produced an increasing number of such injuries, and now that war is causing so many additional cases, it is necessary that we familiarize ourselves with the many problems presented in the proper management of these wounds. Because of his intimate knowledge of the anatomy of these regions and his surgical training, the responsibility should fall largely on the shoulders of the otolaryngologist.

The subject will be illustrated with numerous lantern slides, and the principles of treatment discussed from both the general surgical viewpoint and that of each particular structure.

The Following Are Condensed Schedules of Courses Previously Described, Arranged for Your Convenience by Days and Periods

CONTINUOUS COURSES — OPHTHALMOLOGY

COURSE	SPEAKERS	TITLE	PERIODS USED	Periods Available for Other Subjects
1	Bedell	Medical Ophthalmoscopy	3 PERIODS M-1, T-1, W-1	Six
2	Dunnington	Surgery of the Eyelids and Orbit	3 PERIODS M-3, T-3, W-3	Six
3	Fralick and Crosby	Anatomy of the Orbit	4 PERIODS M-1-2, T-1-2	Five
4	Gailey, et al.	Cataract	3 PERIODS M-1, T-1, W-1	Six
5	Gradle, et al.	Glaucoma	9 PERIODS M-1-2-3, T-1-2-3, W-1-2-3	None
6 or 7	Hardy and Rand	Color Vision Testing	2 PERIODS T-2-3 or W-2-3	Seven
8	Hardy, et al.	Orthoptics	6 PERIODS M-1-2, T-1-2, W-1-2	Three
9	Hughes	Lid Reconstruction	2 PERIODS M-1, T-1	Seven
10 or 11	Lancaster	Anomalous Retinal Correspondence	2 PERIODS T-1-2 or W-2-3	Seven
12	Lyle	Neuro-Ophthalmology	6 PERIODS M-2-3, T-2-3, W-2-3	Three
13	Masters, et al.	Slit Lamp Biomicroscopy	6 PERIODS M-2-3, T-2-3, W-2-3	Three
14	Payne, et al.	Ophthalmology in Aviation	6 PERIODS M-2-3, T-2-3, W-2-3	Three
15	Prangen, et al.	Refraction	9 PERIODS M-1-2-3, T-1-2-3, W-1-2-3	None
16 or 17	Spaeth	Immediate Treatment of Injury of the Lids	2 PERIODS M-1-2 or W-1-2	Seven
18	Sugar	Extraocular Muscles: Diagnosis and Treatment	3 PERIODS M-3, T-3, W-3	Six
19	Theobald, et al.	Pathology	9 PERIODS M-1-2-3, T-1-2-3, W-1-2-3	None
20	White, et al.	Anomalies of the Extra-ocular Muscles	6 PERIODS M-2-3, T-2-3, W-2-3	Three
21	White and Brown	Ocular Motor Anomalies	3 PERIODS M-1, T-1, W-1	Six
22	Woods	Medical Ophthalmology	6 PERIODS M-1-3, T-1-3, W-1-3	Three

CONTINUOUS COURSES — OTOLARYNGOLOGY

50	Anson	Testing of Hearing of School Children	2 PERIODS T-1, W-1	Seven
51	Clerf	Causes of Hoarseness — Treatment	3 PERIODS M-2, T-2, W-2	Six
52	Cullen	Anesthesia in Otolaryngology	2 PERIODS M-1, T-1	Seven
53	Hoerr	Anatomy of Nose, Throat and Ear	3 PERIODS M-3, T-3, W-3	Six
54	Hughson	Audiometric Technics and Fitting of Hearing Aids	3 PERIODS M-2, T-2, W-2	Six
55	Jones	Causes of Dizziness	2 PERIODS M-2, T-2	Seven
56 or 57 or 58	Meltzer	Comparison of Endaural and External Approach in Mastoid Surgery	2 PERIODS M-2-3 or T-2-3 or W-2-3	Seven
59 or 60 or 61	Mueller	Diagnosis of Deafness	2 PERIODS M-2-3 or T-2-3 or W-2-3	Seven
62 or 63	Reger	Audiometry and Hearing Aids	2 PERIODS M-1-2 or T-1-2	Seven
64	Roberts	Biochemistry and Nutrition in Otolaryngology	4 PERIODS T-1-2, W-1-2	Five
65	Shambaugh	Fenestration Operation for Otosclerosis	3 PERIODS M-2, T-2, W-2	Six
66	Skillern	Acute and Chronic Frontal Sinusitis	2 PERIODS M-3, T-3	Seven
67 or 68	Williams	Acute Suppurative Otitis Media and Chronic Mastoiditis	2 PERIODS M-1-2 or W-1-2	Seven

SCHEDULE OF INDIVIDUAL COURSES

Key M-1 1st Period: 8:30 a.m. to 9:30 a.m.

MONDAY

Ophthalmology

Course No.	SPEAKER	SUBJECT
202	Alvis	Acute Corneal Lesions — Diagnosis and Treatment
205	Boeder	Aniseikonia
206	Brown	Ocular Tuberculosis
213	Freeman	Recent Psychological Advances Bearing on Ophthalmology
216	Gittins	Headaches as an Eye, Ear, Nose and Throat Problem
219	Horton	Headaches and Head Pains — Clinical Varieties and Therapeutic Suggestions
220	Joy	Chronic Uveitis Including Sympathetic Ophthalmia
222	Kuhn	Visual Job Analysis, Refraction and Correction for the Job
226	Moore	Management of Intraocular Foreign Body Cases
228	Paton	Keratoplasty

Key M-1 1st Period: 8:30 a.m. to 9:30 a.m.

MONDAY

Otolaryngology

Course No.	SPEAKER	SUBJECT
301	Batson	Anatomic Review of Nose
306	Carmody	Oral Cavity in Health and Disease
308	Fowlkes	Mastoiditis
309	Furstenberg	Reconstruction of Facial Nerve
315	Hansel	Allergy of Nose and Paranasal Sinuses
317	Lempert	Fenestra Nov-Ovalis Operations
318	Lindsay	Meniere's Syndrome
320	McCall	Laryngectomy and Voice Training
322	Mosher	My Milestones
325	Salinger	Deformities of Nasal Septum

Key M-2 2nd Period: 10 a.m. to 11 a.m.

MONDAY

Ophthalmology

Course No.	SPEAKER	SUBJECT
204	Beach	Refinements of Refraction
207	Burch	End Results in Retinal Detachment
209	Cox	Causes, Effects and Remedying of Reading Difficulties
215	Gemeroy	External Diseases of the Eye
232	Sterling	Application of Spectacle Lenses, Theory of Bifocals
234	Town	Contact Lenses

Key M-2 2nd Period: 10 a.m. to 11 a.m.

MONDAY

Otolaryngology

Course No.	SPEAKER	SUBJECT
302	Batson	Anatomic Review of Pharynx and Larynx
304	Beck	Common Disorders in Office and Hospital Practice
305	Campbell	Aviation Otolaryngology
310	Galloway	Laryngeal Emergencies — Bulbar Poliomyelitis
311	Gatewood	X-Ray Interpretation of Sinus Disease
314	Hanley	Anatomy and Anomalies of Temporal Bone
319	Luongo	Anatomy of Fronto-Ethmosphenoidal Region
328	Spake	Intranasal Surgical Anatomy
330	Theobald	Infections of Epipharynx
332	Woodward	Faciomaxillary and Mandibular Fractures

Key M-3 3rd Period: 11:30 a.m. to 12:30 p.m.

MONDAY

Ophthalmology

Course No.	SPEAKER	SUBJECT
201	Adson	Differential Diagnosis and Treatment of Neuralgias of the Face
214	Gamble	Acute Inflammation of the Orbit
218	Heath	Chemotherapy
221	Kronfeld	Gonioscopy
224	Lloyd	Atrophy of the Tapetum with Sclerosis of the Choroidal Vessels
225	Mangold	Types and Preferences in Lens Forms and Fitting of Spectacles
231	Rutherford	Myopia
236	Whalman	Calibration and Application of the Shahan Thermophore in Ophthalmic Conditions

Key M-3 3rd Period: 11:30 a.m. to 12:30 p.m.

MONDAY

Otolaryngology

Course No.	SPEAKER	SUBJECT
303	Batson	Anatomic Review of Ear
307	Dixon	Acute and Chronic Sinusitis
312	Gill	Fractures of Facial Bones
316	Hill	Acute and Chronic Conditions of Maxillary Antrum
321	McMahon	Sinusitis in Children
323	Nash	Functional Diseases of Nose
324	Ryan	Laryngeal and Intrabronchial Tuberculosis
326	Singleton	Operative Procedures on Temporal Bone
327	Snitman	Surgical Anatomy of Neck
329	Straith and Slaughter	Plastic Surgery of Facial Injuries

Key T-1 1st Period: 8:30 a.m. to 9:30 a.m.

TUESDAY

Ophthalmology

Course No.	SPEAKER	SUBJECT
205	Boeder	Aniseikonia
210	Crisp	Cross Cylinders
213	Freeman	Recent Psychological Advances Bearing on Ophthalmology
216	Gittins	Headaches as an Eye, Ear, Nose and Throat Problem
217	Guibor	Value of Orthoptics in Private Practice
219	Horton	Headaches and Head Pains — Clinical Varieties and Therapeutic Suggestions
220	Joy	Chronic Uveitis Including Sympathetic Ophthalmia
222	Kuhn	Visual Job Analysis, Refraction and Correction for the Job
223	Lemoine	Allergy in Ophthalmology
226	Moore	Management of Intraocular Foreign Body Cases
229	Pischel	Retinal Detachment

Key T-1 1st Period: 8:30 a.m. to 9:30 a.m.

TUESDAY

Otolaryngology

Course No.	SPEAKER	SUBJECT
301	Batson	Anatomic Review of Nose
306	Carmody	Oral Cavity in Health and Disease
308	Fowlkes	Mastoiditis
309	Furstenberg	Reconstruction of Facial Nerve
315	Hansel	Allergy of Nose and Paranasal Sinuses
317	Lempert	Fenestra Nov-Ovalis Operations
318	Lindsay	Meniere's Syndrome
320	McCall	Laryngectomy and Voice Training
325	Salinger	Deformities of Nasal Septum

Key T-2 **2nd Period: 10 a.m. to 11 a.m.**

TUESDAY

Ophthalmology

Course No.	SPEAKER	SUBJECT
204	Beach	Refinements of Refraction
207	Burch	End Results in Retinal Detachment
208	Clay	Ophthalmoscopic Classification of Various Hypertensive Diseases
209	Cox	Causes, Effects and Remedying of Reading Difficulties
211	Daily	Physical Therapy in Ophthalmology
212	Davies	Optic Discs — Comparison of Clinical and Pathological Changes
230	Rones	Corneal Dystrophies
233	Stine	Differential Diagnosis and Intraocular Localization of Retinal Detachment
234	Town	Contact Lenses
235	Von der Heydt	Advanced Slit Lamp Microscopy

Key: T-2 **2nd Period: 10 a.m. to 11 a.m.**

TUESDAY

Otolaryngology

Course No.	SPEAKER	SUBJECT
302	Batson	Anatomic Review of Pharynx and Larynx
304	Beck	Common Disorders in Office and Hospital Practice
305	Campbell	Aviation Otolaryngology
310	Galloway	Laryngeal Emergencies – Bulbar Poliomyelitis
311	Gatewood	X-Ray Interpretation of Sinus Disease
319	Luongo	Anatomy of Fronto-Ethmosphenoidal Region
332	Woodward	Faciomaxillary and Mandibular Fractures

Key T-3 **3rd Period: 11:30 a.m. to 12:30 p.m.**

TUESDAY

Ophthalmology

Course No.	SPEAKER	SUBJECT
201	Adson	Differential Diagnosis and Treatment of Neuralgias of the Face
203	Bahn	Fundamentals of Ophthalmic Diagnosis
214	Gamble	Acute Inflammation of the Orbit
218	Heath	Chemotherapy
221	Kronfeld	Gonioscopy
225	Mangold	Types and Preferences in Lens Forms and Fitting of Spectacles
227	Morrison	Malingering
231	Rutherford	Myopia

Key T-3 **3rd Period: 11:30 a.m. to 12:30 p.m.**

TUESDAY

Otolaryngology

Course No.	SPEAKER	SUBJECT
303	Batson	Anatomic Review of Ear
307	Dixon	Acute and Chronic Sinusitis
312	Gill	Fractures of Facial Bones
313	Grove	Anesthesia in Otolaryngology
316	Hill	Acute and Chronic Conditions of Maxillary Antrum
321	McMahon	Sinusitis in Children
323	Nash	Functional Diseases of Nose
324	Ryan	Laryngeal and Intrabronchial Tuberculosis
327	Snitman	Surgical Anatomy of Neck
328	Spake	Intranasal Surgical Anatomy
329	Straith and Slaughter	Plastic Surgery of Facial Injuries

Key W-1 1st Period: 8:30 a.m. to 9:30 a.m.
WEDNESDAY
Ophthalmology

Course No.	SPEAKER	SUBJECT
202	Alvis	Acute Corneal Lesions — Diagnosis and Treatment
205	Boeder	Aniseikonia
206	Brown	Ocular Tuberculosis
210	Crisp	Cross Cylinders
226	Moore	Management of Intraocular Foreign Body Cases
228	Paton	Keratoplasty

Key W-1 1st Period: 8:30 a.m. to 9:30 a.m.
WEDNESDAY
Otolaryngology

Course No.	SPEAKER	SUBJECT
301	Batson	Anatomic Review of Nose
306	Carmody	Oral Cavity in Health and Disease
308	Fowlkes	Mastoiditis
309	Furstenberg	Reconstruction of Facial Nerve
314	Hanley	Anatomy and Anomalies of Temporal Bone
315	Hansel	Allergy of Nose and Paranasal Sinuses
317	Lempert	Fenestra Nov-Ovalis Operations
318	Lindsay	Meniere's Syndrome
320	McCall	Laryngectomy and Voice Training
325	Salinger	Deformities of Nasal Septum
326	Singleton	Operative Procedures on Temporal Bone
330	Theobald	Infections of Epipharynx
331	Tucker	Obstructive Dyspnea and Trachectomy

Key W-2 2nd Period: 10 a.m. to 11 a.m.
WEDNESDAY
Ophthalmology

Course No.	SPEAKER	SUBJECT
208	Clay	Ophthalmoscopic Classification of Various Hypertensive Diseases
212	Davies	Optic Discs — Comparison of Clinical and Pathological Changes
215	Gemeroy	External Diseases of the Eye
223	Lemoine	Allergy in Ophthalmology
229	Pischel	Retinal Detachment
230	Rones	Corneal Dystrophies
232	Sterling	Application of Spectacle Lenses. Theory of Bifocals
233	Stine	Differential Diagnosis and Intraocular Localization of Retinal Detachment
236	Whalman	Calibration and Application of the Shahan Thermophore in Ophthalmic Conditions

Key W-2 2nd Period: 10 a.m. to 11 a.m.
WEDNESDAY
Otolaryngology

Course No.	SPEAKER	SUBJECT
302	Batson	Anatomic Review of Pharynx and Larynx
304	Beck	Common Disorders in Hospital and Office Practice
305	Campbell	Aviation Otolaryngology
319	Luongo	Anatomy of Fronto-Ethmosphenoidal Region
332	Woodward	Faciomaxillary and Mandibular Fractures

Key W-3 3rd Period: 11:30 a.m. to 12:30 p.m.
WEDNESDAY
Ophthalmology

Course No.	SPEAKER	SUBJECT
201	Adson	Differential Diagnosis and Treatment of Neuralgias of the Face
203	Bahn	Fundamentals of Ophthalmic Diagnosis
211	Daily	Physical Therapy in Ophthalmology
217	Guibor	Value of Orthoptics in Private Practice
224	Lloyd	Atrophy of the Tapetum with Sclerosis of the Choroidal Vessels
225	Mangold	Types and Preferences in Lens Forms and Fitting of Spectacles
227	Morrison	Malingering

Key W-3 3rd Period: 11:30 a.m. to 12:30 p.m.
WEDNESDAY
Otolaryngology

Course No.	SPEAKER	SUBJECT
303	Batson	Anatomic Review of Ear
307	Dixon	Acute and Chronic Sinusitis
312	Gill	Fractures of Facial Bones
313	Grove	Anesthesia in Otolaryngology
316	Hill	Acute and Chronic Conditions of Maxillary Antrum
323	Nash	Functional Diseases of Nose
327	Snitman	Surgical Anatomy of Neck
329	Straith and Slaughter	Plastic Surgery of Facial Injuries

Trial Notes

It may be helpful in orienting selections if you use this trial schedule prior to filling in the order sheet.

MORNING HOURS

	M (Monday)	T (Tuesday)	W (Wednesday)
1st Period	Course No.		
	Instructor		
2nd Period	Course No.		
	Instructor		
3rd Period	Course No.		
	Instructor		

BUSINESS MEETING

Grand Ball Room — 8 p.m.
WEDNESDAY, OCTOBER 11

Reports of Standing Committees will be read and later published in the November-December issue of the TRANSACTIONS. The Senior Member of the Council will report Council action upon Committee recommendations because changes in Committee reports as recommended by the Council, or new business originating from the floor, must be referred to the Council for action.

ORDER OF BUSINESS

1. Call to order.
2. Report of Executive Secretary-Treasurer.
3. Report of Necrologist.
4. Report of Editor of TRANSACTIONS.
5. Committee reports.
6. Report of American Board of Ophthalmology.
7. Report of American Board of Otolaryngology.
8. Unfinished business.
9. New business.
10. Election of Fellows.
11. Report of the Senior Member of the Council.
12. Election of officers (Senior Member of Council report).

1944 Candidates

Objections to the election of a candidate must be filed with the Executive Secretary previous to Sunday, October 8, 1944.

CANDIDATES CERTIFICATED BY BOARDS

Ophthalmology (Op.)
Otolaryngology (ALR)
Plastic Surgery (Pl.S.)

Aageson, Carl W., 1 South Pinckney St., Madison, Wis., Op.

Abbott, Henry B., 423 Main St., Geneva, N. Y., Op.

Alliband, George T., Lt. (MC) USNR, U. S. Naval Air Station Dispensary, Corpus Christi, Texas, Op.

Alpert, David R., Lt. (MC), 28 Verndale St., Brookline, Mass., Op.

Armstrong, Richard C., Capt. (MC), Station Hospital, Peterson Field, Colorado Springs, Colo., Op.

Ash, Arthur F., 710 Boulevard East, Weehawken, N. J., ALR.

Baker, Frederick A., 1102-3 Peoples State Bldg., Pontiac, Mich., Op.

Bauer, Arthur S., Gallinger Hospital, Washington 3, D. C., ALR.

Beall, John G., University Hospital, Ann Arbor, Mich., Op.

Beauchamp, Mark L., Capt. (MC), 2306 5th Avenue, Los Angeles, Calif., ALR.

Benson, Kenneth H., Major (MC), Station Hospital, Luke Field, Phoenix, Ariz., ALR.

Berger, Meyer D., 681 Ocean Ave., Brooklyn 26, N. Y., ALR.

Bernstein, David, Major (MC), Veterans Administration, Tucson, Ariz., ALR.

Blackford, Roger W., 503 Hume-Mansur Bldg., Indianapolis 4, Ind., ALR.

Boyd, Harold M. E., Lt (MC) Santa Ana Army Air Base, Santa Ana, Calif., ALR.

Brandon, James R., 800 1st National Bank Bldg., Canton, Ohio, ALR.

Breisacher, Carl F., Dartmouth Eye Institute, Hanover, N. H., Op.

Brodnitz, Friedrich S., 667 Madison Ave., New York, N. Y., ALR.

Brown, Harold W., 15 Park Ave., New York, N. Y., Op.

Burtoff, Samuel, Lt. (MC) 4410 16th Street N. W., Washington, D. C., ALR.

Chace, Robert R., 635 W. 165th St., New York 32, N. Y., Op.

Chamberlain, Webb P., Jr., Capt. (MC), A.P.O. 322, c/o Postmaster, San Francisco, Calif., Op.

Chamlin, Max, 1840 Grand Concourse, New York, N. Y., Op.

Christensen, Eugene L., 727 W. 7th St., Los Angeles 14, Calif., Op.

Clements, F. H., 806 Medical Arts Bldg., Birmingham, Ala., Op.

Colson, Z. William, 301 Essex Street, Lawrence, Mass., Op.

Cox, J. Robert, Lt. (MC), Army and Navy General Hospital, Hot Springs, Ark., Op.

Crawford, James Harry, Buffalo, Minn., Op.

Currier, W. D., 65 N. Madison Ave., Pasadena 1, Calif., ALR.

Daly, John F., Capt. (MC), AAF Regional Hospital No. 1, Unit N, Miami Beach, Fla., ALR.

Dattner, Herman B., Major (MC) Sqd. H, 330th AAF Base Unit, Greenville Army Air Base, Greenville, S. C., ALR.

DeBord, Bert A., Capt. (MC) Station Hospital, Frederick Army Air Field, Frederick, Okla., ALR.

deRoetth, Andrew F., 508 Old National Bank Bldg., Spokane 8, Wash., Op.

Dickey, C. Allen, 450 Sutter St., San Francisco 8, Calif., Op.

Diehl, James E., 813 Wainwright Bldg., Norfolk, Va., ALR.

Dineen, John B., 1537 Main St., Springfield, Mass., ALR.

Dolph, Chancey H., 317 Main, Baytown, Texas, ALR.

Doust, Alfred W., Major (MC), Station Hospital, Fort Jay, New York 4, N. Y., ALR.

Ehrenfeld, Edward, 185 Lexington Ave., Passaic, N. J., Op.

Ellis, Van M., 1530 Locust St., Philadelphia 2, Pa., Op.

Emenhiser, Lee K., Lt. Col. (MC), Brooke General Hospital, Ft. Sam Houston, Texas, ALR.

Erich, John B., Mayo Clinic, Rochester, Minn., Pl.S.

Evans, Samuel D., 355 5th Ave., Pittsburgh 22, Pa., Op.

Fischer, Louise, Medical Towers, 31 Lincoln Park, Newark 2, N. J., ALR.

Foote, C. M., 412 N. Hastings Ave., Hastings, Neb., Op.

Ford, Harry C., Major (MC), A.P.O. 9907, c/o Postmaster, New York, N. Y., ALR.

Fortenberry, L. S., Capt. (MC), Brooks Field, San Antonio, Texas, ALR.

Freeman, Marvin S., Capt. (MC), A.P.O. 63, c/o Postmaster, New York, N. Y., ALR.

Friedman, Bernard B., 916 Jones Bldg., Corpus Christi, Texas, Op.

Furlong, Frank C., 713 Union St., Schenectady, N. Y., ALR.

Gaillard, Milton T., 5 Park Ave., Baldwin, N. Y., Op.

Gettes, Charles N., 481 Beacon St., Boston 15, Mass., ALR.

Gibb, W. Blake, 1515 State Street, Santa Barbara, Calif., Op.

Girard, Paul H., 258 Genesee St., Utica, N. Y., Op.

Glasgow, S. Condit, 8 East Alisal St., Salinas, Calif., ALR.

Glauber, Jerome J., Capt. (MC), Station Hospital, Drew Field, Tampa, Fla., ALR.

Godwin, Dean E., 117 E. 8th St., Long Beach 2, Calif., ALR.

Gordon, Benjamin L., Professional Arts Bldg., Atlantic City, N. J., Op.

Green, Stanley L., 129 W. Lexington, Independence, Mo., Op.

Gressette, James H., Box 2467, Roanoke 10, Va., ALR.

Grossmann, Erwin E., Major (MC), Station Hospital, Camp Maxey, Texas, Op.

Gunderson, Ernest O., Major (MC), Station Hospital, Ft. Lewis, Wash., Op.

Hall, Donald S., Capt. (MC), Station Hospital, Garden City A.A.F., Garden City, Kan., OALR.

Hallaron, John J., Capt. (MC), Army Air Base Station Hospital, Great Falls, Mont., ALR.

Harris, Louise D., Lt. (MC), Oakland Regional Hosp., Oakland 14, Calif., Op.

Harris, Morris, Capt. (MC), Station Hospital, Ft. H. G. Wright, N. Y., Op.

Harrison, Glen H., 307 Washington St., Waukegan, Ill., Op.

Hart, Noah Leon, Capt. (MC), Station Hospital, Army Air Base, Harding Field, La., ALR.

Hartman, Deane C., 727 W. 7th St., Los Angeles 14, Calif., Op.

Haughton, Thomas J., Major (RCAMC), No. 1 Canadian General Hospital, Canadian Army, Cen. Med. Forces, ALR.

Hausmann, Gertrude S., 629 16th St., Denver 2, Colo., Op.

Haven, Walter K., 1009 Nicollet Ave., Minneapolis 2, Minn., OALR.

Hawn, Hugh W., Capt. (MC), A.P.O. 557, c/o Postmaster, New York, N. Y., Op.

Heckert, Frank B., 1105 Bank of Lansing Bldg., Lansing, Mich., Op.

Hersh, Joseph H., Capt. (MC), Station Hospital, Camp Stoneman, Calif., ALR.

Hewitt, Wright P., 51 Brattle Street, Cambridge 38, Mass., ALR.

Hoitash, Frederick J., 304 Fifth Ave. Arcade, Huntington 13, W. Va., Op.

Hopp, Eugene S., Major (MC), A.P.O. 547, c/o Postmaster, New York, N. Y., ALR.

Huggins, Harrison D., 12 Whipps Bldg., Kalispell, Mont., ALR.

Huston, Marshall D., Capt. (MC), Station Hospital, Camp Bowie, Brownwood, Texas, ALR.

James, James Edward, 128 W. Fourth St., Bethlehem, Pa., ALR.

Johnson, Fordyce A. H., Capt. (MC), Pasadena Regional Hosp., Pasadena 2, Calif., ALR.

Jones, Arthur C., 518 Eastman Bldg., Boise, Idaho, ALR.

Jones, Emmett L., Jr., Major (MC), Station Hospital, Army Air Field, Ardmore, Okla., ALR.

Jones, William Moses, 5254 Michigan Ave., Chicago, Ill., Op.

Joseph, N. K., 40-14th Street, Wheeling, W. Va., ALR.

Kaiser, Robert F., Capt. (MC), Gorgas Hospital, Ancon, Canal Zone, ALR.

Kelley, Samuel F., 47 E. 61st St., New York, N. Y., ALR.

Kelly, Vincent J., Capt. (MC) Station Hospital, Ellington Field, Texas, OALR.

Keskey, George I., Rose Bldg., Marquette, Mich., ALR.

Kieler, Charles B., M. I. B. Hospital, Cooperstown, N. Y., ALR.

King, Charles M., Jr., Capt. (MC), A.P.O. 526, c/o Postmaster, New York, N. Y., Op.

King, Raymond H., Lt. Comdr. (MC), U. S. Naval Hospital, Jacksonville, Fla., ALR.

Kingston, Louis C., Capt. (MC), Tilton General Hospital, Fort Dix, N. J., ALR.

Kleiman, Bernard S., 3318 Bancroft Road, Baltimore, Md., ALR.

Knudsen, Arnold F., 419 Bay Ridge Parkway, Brooklyn 9, N. Y., ALR.

Koke, Martin P., 233 A Street, San Diego, Calif., Op.

Kupp, John H., 582 Delaware Ave., Palmerton, Pa., ALR.

Kutscher, Charles F., 520 Medical Arts Bldg., Pittsburgh 13, Pa., Op.

Lance, Verne L., 1031 Quatrier St., Charleston, W. Va., ALR.

Langworthy, Henry G., 394 W. 10th St., Dubuque, Iowa, ALR.

Lanson, F. Arthur, 646 Washington Road, Mt. Lebanon, Pa., ALR.

Larmore, Joseph L., Capt. (MC), Station Hospital, Camp Gruber, Okla., ALR.

Leath, MacLean B., 511 N. Main St., High Point, N. C., ALR.

Lenholt, Eric H., Capt. (MC), Station Hospital, Spence Field, Moultrie, Ga., ALR.

Leonard, J. C., Jr., 3½ E. First Ave., Lexington, N. C., ALR.

Lieberman, Alfred T., Capt. (MC), 553rd AAF Base Unit, AAF Station Hospital, R.A.A.F., Romulus, Mich., ALR.

Lieberthal, Paul R., 104 S. Suffolk, Ironwood, Mich., Op.

Loutfallah, Michel, 1826 State Street, Santa Barbara, Calif., Op.

Lowenstein, Harold H., Capt. (MC), Station Hospital, Camp Sutton, N. C., Op.

Lowy, Karl, 35 Chestnut Street, Rochester, N. Y., ALR.

Lubart, Joseph, 310 West 86th St., New York, N. Y., ALR.

Lyman, Edward H., Capt. (MC), A.A.F. Regional Station Hospital, Langley Field, Va., ALR.

Lynam, Patrick J., Lt. Comdr. (MC), 198 Maple Street, Malden, Mass., ALR.

McDaniel, R. L., 211 St. James Bldg., Jacksonville, Fla., Op.

McLaughlin, Ralph S., 805 Atlas Bldg., Charleston, W. Va., Op.

MacMillan, Karl D., Lt. Col. (MC), Woodrow Wilson General Hospital, Staunton, Va., Op.

Marchman, Oscar M., Jr., Major (MC), Station Hospital, Hobbs A.A.F., Hobbs, N. M., Op.

Matusow, Meyer, 1075 West Farms Road, New York, N. Y., Op.

May, James W., Lt. (MC) USNR, U. S. Naval Dispensary, Navy Department, Bellevue, Washington 25, D. C., Op.

Miller, Daniel, 20 Charlesgate West, Boston 15, Mass., ALR.

Miller, M. S., Major (MC), 206 11th Ave. N. E., Calgary, Alberta, Canada, ALR.

Mogan, Richard F., 523 West 6th St., Los Angeles 14, Calif., ALR.

Monahan, Richard O'B., Box 2467, Roanoke 10, Va., ALR.

Morgan, George E., Lt. (MC) Station Hospital, Unit II, Marsh Field, Riverside, Calif., Op.

Morrison, George S., 308 W. 2nd St., Roswell, N. M., ALR.

Mosher, W. F., 34 N. Ash Street, Ventura, Calif., ALR.

Mountjoy, Philip S., Capt. (MC), School of Aviation Medicine, Randolph Field, Texas, ALR.

Nacey, L. J., 221 Alexander, Rochester, N. Y., ALR.

Nelson, Fritz, 1121 North Tejon St., Colorado Springs, Colorado, Op.

Newby, Harley D., 619½ Main St., Rapid City, S. D., ALR.

Newman, Saul C., Major (MC), Nichols General Hospital, Louisville 2, Ky., ALR.

Oakes, Milton C., Major (MC), 61st General Hospital, Camp Grant, Ill., Op.

Ogden, Frederick W., Capt. (MC), A.A.F. School of Aviation Medicine, Randolph Field, Texas, ALR.

Olmsted, K. Elizabeth Pierce, 568 Lafayette Ave., Buffalo 13, N. Y., Op.
Ornston, D. G., 36 Carpenter Lane, Philadelphia 19, Pa., ALR.
Ostrow, Irving, Capt. (MC), 1570 S. U., Camp Breckenridge, Ky., ALR.
Owens, Harold, 1136 W. 6th Street, Los Angeles 14, Calif., ALR.
Palevsky, Samuel N., Major (MC), Hoff General Hospital, Santa Barbara, Calif., ALR.
Pannabecker, Charles L., University Hospital, Ann Arbor, Mich., Op.
Patterson, Sam H., 125 15th St., Toledo 2, Ohio, Op.
Payne, Frank, 635 W. 165th St., New York 32, N. Y., Op.
Peterson, Roy L., 518 Eastman Bldg., Boise, Idaho, ALR.
Phillips, Earl H., Capt. (MC), Hoff General Hospital, Santa Barbara, Calif., ALR.
Pickard, J. C., 1200 Main St., Dubuque, Iowa, ALR.
Pitman, Louis K., Major (MC), A. A. F. O. R. D., Greensboro, N. C., ALR.
Polan, Charles M., Lt. (MC), Station Hospital, Camp Luna, N. M., Op.
Potts, Charles R., 1304 Walker Ave., Houston 2, Texas, Op.
Poulin, James E., Professional Bldg., Waterville, Maine, ALR.
Powell, A. C., Lt. Comdr. (MC), Dispensary, Naval Air Station, Whidby Island, ALR.
Preefer, Raymond R., Major (MC), Billings General Hospital, Ft. Ben Harrison, Ind., Op.
Proctor, Donald F., 6 East Eager St., Baltimore 2, Md., ALR.
Prohovnik, Julius, 9 South Kedzie Ave., Chicago, Ill., ALR.
Quereau, James VanDyke, 206 N. 5th Street, Reading, Pa., Op.
Ramm, Carl H., 508 Main Street, Bennington, Vt., Op.
Ray, George H., New York Eye and Ear Infirmary, New York, N. Y., Op.
Ray, Harold E., 135 East Main St., Xenia, Ohio, Op.
Reeves, J. Norman, 121 Main St., Western Port, M.D., ALR
Rigby, Ralph G., 700 N. Michigan Ave., Chicago, Ill., ALR.
Rittenhouse, Emory A., Major (MC), Station Hospital, Fort Myer, Va., ALR.
Ruggeri, Charles, Jr., 1120 Boston Bldg., Salt Lake City 1, Utah, Op.
Russell, Ralph E., Capt. (MC), A.A.F. M.S.T.S., Robins Field, Ga., Op.
Ryan, Michael J., Capt. (MC), 44th Evacuation Hospital, Fort Dix, N. J., ALR.
Schattner, Alfred, 115 E. 61st St., New York, N. Y., ALR.
Schutz, Sigmund, Capt. (MC), Station Hospital, Dover Army Air Base, Dover, Del., Op.
Senecal, A. L., 1 Monument Square, Beverly, Mass., ALR.
Simons, Harold N., Major (MC), Rhoads General Hospital, Utica, N. Y., Op.
Sklarow, Louis, Bank Bldg., Gowanda, N. Y., ALR.
Smedal, Erling A., Capt. (MC), A.A.F. Regional Station Hospital, Langley Field, Va., Op.
Smith, Dennis V., 110 Pine Street, Long Beach, Calif., Op.
Smith, Mason, Lt. (j.g), (MC) USNR, U. S. Naval Hospital, New Orleans, La., Op.
Smith, Maynard P., 206 Professional Bldg., Richmond, Va., ALR.
Sneller, Charles D., 320 Jefferson Bldg., Peoria, Ill., ALR.
Snow, William J., 1215 Walker Ave., Houston, Texas, ALR.
Soldineer, Marshall Y., Capt. (MC), Station Hospital, Fort Leonard Wood, Mo., ALR.
Spain, M. L., 926 Main St., Rapid City, S. D., ALR.
Spear, Dean, 720 North Michigan, Chicago 11, Ill., Op.
Steffens, Lincoln F., Lt. Col. (MC), Station Hospital, Camp Chaffee, Ark., Op.
Stephens, H. Frederick, Lt. Comdr. (MC), USNR, 195 Thayer Street, Providence, R. I., Op.
Stewart, Marshall B., Major (MC), Brooke General Hospital, Fort Sam Houston, San Antonio, Texas, ALR.
Stow, M. Noel, 635 W. 165th Street, New York 32, N. Y., Op.
Strange, Russell Hope, 117 S. College Ave., Mt. Pleasant, Mich., ALR.
Streicher, Carl J., 610 North Market, Canton, Ohio, Op.
Strickland, Horace G., Lt. Comdr. (MC), USNR, U. S. Naval Dispensary, Navy Department, Washington 25, D. C., Op.
TenEyck, T. G., Major (MC) Station Hospital, Ft. Lewis, Wash., ALR.
Trapasso, Anthony J., Lt. (MC), Station Hospital, Army Air Field, Patterson Field, Ohio, ALR.
Truex, Edward H., Jr., Capt. (MC), Deshon General Hospital, Butler, Pa., ALR.
Uhler, Ella Messinger, 104 North 8th St., Allentown, Pa., Op.

Urban, Joseph T., Lt. Comdr. (MC), USNR, U. S. Naval Dispensary, Navy Department, Washington 25, D. C., ALR.

Urist, Martin J., Center and Phoenix Sts., South Haven, Mich., Op.

Van Bergen, Thomas M., Capt. (MC), Station Hospital, Camp Cooke., Calif., Op.

Viviani, John J., 180 Suydam Street, Brooklyn 21, N. Y., ALR.

Walker, Glenn L., Lt. Col. (MC), Thayer General Hospital, Nashville, Tenn., Op.

Walsh, Frank B., Johns Hopkins Hospital, Baltimore 5, Md., Op.

Weiss, Sidney, Lt. (MC), Station Hospital, Camp Crowder, Mo., Op.

Wetzel, R. J., 301 Broadway, Hanover, Pa., ALR.

Wilner, Arthur S., 225 West State Street, Trenton, N. J., ALR.

Wood, Maynard A., 128 N. 13th Street, Lincoln, Nebr., Op.

Wynn, Sidney K., Lt. (MC), Regional Hospital, Ft. Francis E. Warren, Wyo., ALR.

Yasuna, Elton R., Lt. (MC), 340 Walnut Ave., Roxbury, Mass., Op.

CANDIDATES NOT CERTIFICATED
(Eligible for Membership After Board Certification)

Aguero, Alexis, San Jose, Costa Rica

Carrozza, Natale S., 108 Vanderbilt St., Brooklyn 18, N. Y.

Collins, John A., 406-8 Union Savings & Trust Bldg., Warren, Ohio

deRezende, Nilson T., Esteves Jr. 68, Rio, Brazil, S. A.

Feiman, Edward M., 212 First National Bank Bldg., Canton 2, Ohio.

Hargett, Edgar R., 4 West Main Street, Springfield, Ohio

Levey, Mark R., 830 Tegler Building, Edmonton, Canada.

Munoz-MacCormick, Carlos E., Monterrey Building, Park Terrace, Santurce, Puerto Rico

Perry, James S., McCartney Drug Store, Bryan, Texas

Roszell, Leo H., 131 Fulton Avenue, Hempstead, L. I., New York.

Rowland, Alan L., 1031 Bank of America Building, San Diego 1, Calif.

Seelye, Norman Lee, 36 North Ayer St., Harvard, Ill.

Vermillion, Dale D., Goodland, Kan.

Winston, Samuel H., Reeves Bank Bldg., Dover, Ohio.

Zarbaugh, Guy F. (U. S. Navy Reserve), c/o Fleet Post Office, San Francisco, Calif.

Necrology

IN MEMORIAM

William Bean Anderson, Brownwood, Texas

Max Bakst, Brooklyn, N. Y.

Henry Bush Beeson, Racine, Wis.

Will Otto Bell, Seattle, Wash.

Edmond E. Blaauw, Buffalo, N. Y.

Oliver Preston Bourbon, Los Angeles, Calif.

Charles Hermann Brobst, Peoria, Ill.

Morrow Duncan Brown, Denver, Colo.

Ralph Stephen Chappell, Indianapolis, Ind.

William Morgan Chew, New York, N. Y.

William Franklin Clevenger, Indianapolis, Ind.

John Hamilton Cooper, Massillon, Ohio

William Thornwall Davis, Washington, D. C.

Lee Wallace Dean, St. Louis, Mo.

Zenas Horace Ellis, New York, N. Y.

Robert J. Ferguson, New Haven, Conn.

Carl Fisher, Los Angeles, Calif.

George Foster Fiske, Chicago, Ill.

Arthur Emile Gadbois, Norfolk, Neb.

Sanford Robinson Gifford, Chicago, Ill.

Herbert Maskell Goddard, Philadelphia, Pa.

George Robert Gowen, Walla Walla, Wash.

John Milton Griscom, Philadelphia, Pa.

Israel Grushlaw, New York, N. Y.

William Carleton Harris, Cincinnati, Ohio

Raymond Willard Hawkins, Rochester, N. Y.

Ernest Reed Hirst, Camden, N. J.

Samuel Iglauer, Cincinnati, Ohio

Harry Morgan Ivins, Santa Cruz, Calif.

George Ernest Johnson, Philadelphia, Pa.

George Lindsay Johnson, Durban, Natal, Africa

Wilson Johnston, Portland, Ore.

Charles Curtis Jones, Cincinnati, Ohio

Carl Koller, New York, N. Y.

William Andrew Krieger, Poughkeepsie, N. Y.

Max Lubman, New York, N. Y.

Charles Henry May, New York, N. Y.

Myron Firth Metzenbaum, Cleveland, Ohio

Joseph Edward Miller, Los Angeles, Calif.

John W. Millette, Dayton, Ohio

Millen Alexander Nickle, Clearwater, Fla.

William Wilson Pearson, Des Moines, Iowa

Reginald Stirling Pentecost, Toronto, Ontario, Canada

Adolph O. Pfingst, Louisville, Ky.

Grover Hewitt Poos, Palm Springs, Calif.

Edward Hyndshaw Porter, Tiffin, Ohio

Paul Judd Sartain, Philadelphia, Pa.

Carl Francis Schaub, Chicago, Ill.

Frank L. Secoy, Sioux City, Iowa

William Alfred Shoemaker, St. Louis, Mo.

Raphael Silva, Mexico City, Mexico

Otis Franklin Simonds, Cleveland, Ohio

Raymond Duane Sleight, Battle Creek, Mich.

Louis Sam Smith, Brooklyn, N. Y.

Harmon Lamontte Stanton, Evansville, Ind.

Charles Midwood Stiles, Philadelphia, Pa.

George Hamilton Walker, Winona, Minn.

WILLIAM BEAN ANDERSON

Dr. Anderson was born February 26, 1863, in Columbiana, Ala., the son of Dr. Daniel McKaskle and Martha Elizabeth (Bean) Anderson. In 1866, he came to Anderson County, Texas, with his parents. His academic education was obtained in Waco University (now Baylor University). His medical education was received in Vanderbilt University, Nashville, Tenn., from which he was graduated in 1888. He spent two and one-half years practicing medicine in Williamson County, and three years in Runnels County before attending Tulane University, New Orleans, where in 1894, he received his second M.D. degree. After graduation from Tulane University, Dr. Anderson settled in Brownwood, Texas. His death brought to an end almost fifty years of professional service to this community. During his years of practice he kept abreast of the advancement of medicine by graduate work at the Chicago Post-graduate School, New York Polyclinic, and the New York Eye and Ear Infirmary where he received special training in eye, ear, nose and throat. He limited his practice to these specialties in 1903.

Dr. Anderson was a charter member of the Brown County Medical Society and later a member of other county and state societies. A fellow of the American Medical Association and the American College of Surgeons and an organizer of the Fourth District Medical Society of Texas of which he was twice elected president, Dr. Anderson was also a Fellow of the American Academy of Ophthalmology and Otolaryngology to which he was elected in 1913. He was a charter member of the Texas Society of Ophthalmology and Otolaryngology. He was one of the founders of the Medical Arts Hospital, Brownwood, which he served as a director and as a member of the staff since its organization in 1927. He was chief of the eye, ear, nose and throat department of that institution at the time of his death.

Dr. Anderson was a deacon in the Baptist Church for many years. He was also a trustee of Howard Payne College, Brownwood, for many years, and a member of the Masonic lodge.

In 1888, Dr. Anderson was married to Miss Frances Elizabeth Lewis who survives him. His hobby, the collection of rare rocks and minerals, was also enjoyed by Mrs. Anderson and their private collection is now one of the finest in the South.

MAX BAKST

Dr. Bakst was born in 1886 in Russia, where he also received his preliminary education. He obtained his medical degree at the Long Island College of Medicine, Brooklyn, N. Y., in 1911. Postgraduate work was taken at Temple University, Philadelphia. In 1912 he began his practice of otolaryngology in Providence. Three years later he moved to Brooklyn. He was associate attending physician in otolaryngology at Beth Moses Hospital there for several years.

Dr. Bakst was a fellow of the American Medical Association and the American Academy of Ophthalmology and Otolaryngology. He also belonged to local medical societies. Outside of his medical practice, he was interested in painting.

Besides his wife, Dr. Bakst is survived by a son and a daughter. His death occurred November 10, 1943.

HENRY BUSH BEESON

Dr. Beeson, 62 year old Racine, Wis., physician, died at his home March 4, 1944. Born in Fond du Lac, he graduated from Hahnemann Medical College and Hospital, Chicago, in 1908 and from Northwestern University Medical School, Chicago, in 1912. He studied postgraduate work at the Illinois Eye and Ear Infirmary.

Until 1934, Dr. Beeson practiced in Colorado Springs, Colo. During World War I, he served in France as a captain of the 142nd division. In Racine he was on the staff of the Sunny Rest Sanatorium, a member of the senior staff and chief of the department of ophthalmology and otolaryngology at St. Mary's Hospital, and a member of the associate staff of St. Luke's Hospital.

Besides being a Fellow of the American Academy of Ophthalmology and Otolaryngology and the American Medical Association, he belonged to the Milwaukee Society of Otolaryngology, which he served as vice president in 1940-41. He also belonged to the Racine County Medical Society and the State Medical Society of Wisconsin.

His wife and three sons survive him.

WILL OTTO BELL

Dr. Bell was born in Anoka, Minn., in 1876. At the age of nine he moved with his mother to Seattle, Wash. He graduated from Shattuck Military Academy, Fairbault, Minn., and in 1907 from the

University of Illinois College of Medicine.

His extensive postgraduate study took him to Vienna, Budapest, London, Innsbruck and Basel. He specialized in ophthalmology and became widely known for his expert work with the slit-lamp.

In addition to membership in several state and regional medical societies, Dr. Bell was a Fellow of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association, and the American College of Surgeons. His fraternity was Beta Theta Pi and he also belonged to the Washington Athletic Club and the Eight Millimeter Club.

Dr. Bell passed away February 3, 1944. Surviving are his wife, Louise; a son, Alan, at Johns Hopkins Medical College; and a daughter, Ola Louise.

EDMOND E. BLAAUW

Dr. Blaauw was born in Arnhem, Holland. When quite young his family moved to Ryswyk near The Hague where he went first to school. At the age of 19 he began the study of medicine at the University of Amsterdam. He studied also at Freiburg, Paris, and Lyons. In Paris two of his teachers were Landolt and DeWecker. For two years before he was graduated he was nominally assistant to Professor Gunning in the Amsterdam Eye Hospital but actually he was in charge most of the time. His thesis for his medical degree was on the subject of trachoma. After obtaining his degree he married Marianne de Lange in Holland. They came at once to America, settling in Buffalo where he practiced his specialty until his death.

Dr. Blaauw never lost his intellectual loyalty to the European institutions. He made several trips abroad to attend medical meetings — once a congress at Heidelberg, another time at Naples, and again at Rome. He made a trip around the world, visiting the ophthalmologic clinics and the centers of art and learning in India, China, Japan and elsewhere. He acquired on this journey many publications on Oriental philosophy and art. He was keen in his appreciation of the beautiful in nature and its reproduction on canvas. He came by this naturally for his father was a painter of decided merit.

Dr. Blaauw is perhaps best remembered as one who was outstanding in his knowledge of medical literature and especially that relating to ophthalmology. He had a library which so expanded through the years that little room was left in his home for anything else. He read incessantly. He had a most retentive memory. Those who knew him can testify to the remarkable way he could delve into that memory and bring out something pertinent to the subject under discussion. To his Buffalo colleagues it was, "Let's ask Blaauw," and he would, without hesitation, bring a pamphlet, a journal, or textbook out for an answer by an authority.

His reading and his work took so much time that little was left for play. He was an ardent lover of music and played the piano with skill.

Dr. Blaauw could be called an enthusiast in medicine. Asked a medical question, he was at once stirred to mental and physical activity. His patients were cases, all interesting as such. He thought of their physical condition, not their pocketbooks, and so he was not successful in the business sense. He published about twenty-five articles.

He was associated with the Buffalo Deaconess Hospital, the Buffalo Eye and Ear Infirmary, and the Charity Eye Hospital. He was a member of the County Society, the Buffalo Academy of Medicine, the American Medical Association, the American Academy of Ophthalmology and Otolaryngology, and of several European societies.

He developed trouble with his heart and died suddenly during the night of December 4th, 1943. Surviving is his wife.

OLIVER PRESTON BOURBON

Dr. Bourbon passed away on April 24, 1944, at the age of 80 years. He was born in Wisconsin on October 15, 1863, and received his degree of doctor of medicine in 1908 from the Kansas City Hahnemann Medical College. Subsequently he did work at the New York Eye and Ear Infirmary and St. Bartholomew's Hospital in New York City. For fourteen years he practiced in Kansas City, Mo., and during that time was member of the Jackson County and Missouri State Medical Association, and served on the staff of Childrens Mercy Hospital of Kansas City. He was a member of the ear, nose and throat staff of the Swope Settlement Dispensary and on the consulting staff of the Missouri Commission of the Blind. In 1924 he came to Los Angeles, where he has practiced continuously. He formerly was instructor in ophthalmology at the Col-

lege of Medical Evangelists, and among his reprints are those from the Journal of the American Medical Association and the Archives of Otolaryngology.

CHARLES HERMANN BROBST

Dr. Brobst was born January 21, 1864, in Stinesville, Pa. His early education was procured there and in 1888 he received the degree of doctor of medicine from the Medico-Chirurgical College of Philadelphia. After graduation he took postgraduate work at Johns-Hopkins in Baltimore and later continued his studies in Queens Hospital, London, England.

Dr. Brobst began his practice of medicine in Pennsylvania, later moving to Chicago, and then settling in Peoria, Ill., in April of 1893, where he became a member of the staff of the Proctor Hospital, an association which was maintained for forty-five years. For many years he lectured to the nurses in that institution.

Dr. Brobst was a Life Fellow of the American Academy of Ophthalmology and Otolarynogology having been elected to membership in 1906. He was also a fellow of the American College of Surgeons and a member of state and local medical societies. Outside of his professional duties, Dr. Brobst was active in local affairs. He was a life member of the Knights of Constantine and the Mohammed Temple Shrine. He was a member of the First Federated Church.

His death occurred November 25, 1943. Surviving are his daughter, Mrs. Dorothy Brobst Partee, and a son, Captain Charles D. Brobst of the medical corps, now serving overseas.

MORROW DUNCAN BROWN

Dr. Brown was born February 20, 1878, in Cedar Rapids, Iowa. He attended high school in Chicago, and was graduated from the medical and literary departments of Northwestern University, receiving his doctor of medicine degree in 1900. He first practiced in Chicago after the death of his father there.

In 1901, Dr. Brown moved to Chama, N. M., where he was physician for the Denver and Rio Grande Western railroad and a large lumber company. In 1902, he married Elizabeth Putnam, great-granddaughter of General Israel Putnam of Revolutionary War fame. He was licensed to practice in Colorado in 1907.

Dr. Brown was a major in the army medical corps in World War I. Later he entered private practice as an ear, nose and throat specialist which he continued until his death. He was on the staffs of the Mercy, Presbyterian, St. Josephs, Childrens' and St. Anthony Hospitals and the Porter Sanatorium. He was also head of the ear, nose and throat department of the Lutheran Sanatorium for twenty-five years.

Dr. Brown became a Fellow of the American Academy of Ophthalmology and Otolaryngology in 1920. He was also a fellow of the American Medical Association, a member of state and county medical societies, the Colorado Society for Crippled Children and the American Laryngological, Rhinological and Otological Society. Dr. Brown was a Mason and a member of the American Legion, the Denver Athletic Club and the Lakewood Country Club. He attended the United Presbyterian Church.

Dr. Brown died at his home in Denver, July 1, 1943, two weeks after the death of his wife. He is survived by a daughter, Miss Grettamae Brown, two sisters and a brother.

RALPH STEPHEN CHAPPELL

Dr. Chappell was born February 12, 1880, in Petersburg, Ind. He attended Vincennes Military Academy and received the degree of doctor of medicine from the Medical College of Indiana in 1904. Upon graduation he began practice in Indianapolis. His work was limited to diseases of the eye, nose and throat. Dr. Chappell undertook graduate study at Harvard in 1919 and in Vienna in 1921.

He was assistant professor of otolaryngology at the Indiana University School of Medicine. He was a founder and the first president of the Indianapolis League for the Hard of Hearing. He served as president of this organization for seven years. He was also consulting otolaryngologist and secretary of the board of trustees of the Indiana School for the Deaf for the past twelve years. From 1932 to 1938, Dr. Chappell served as chief of the ear, nose and throat staff at the City Hospital. At the time of his death he was on the staffs of the Methodist and St. Vincent Hospitals.

Dr. Chappell was a member of the Marion County Medical Society, the Indianapolis Ophthalmological and Otolaryngological Society, and the Indiana

Academy of Ophthalmology and Otolaryngology. He was a fellow of the American Medical Society. In 1925 he was elected to Fellowship in the American Academy of Ophthalmology and Otolaryngology.

Dr. Chappell died March 12, 1944, leaving his wife and a daughter.

WILLIAM MORGAN CHEW

Lieutenant Commander Chew, M. C., U.S.N.R., was born in Waynesboro, Va., in 1904. He attended Fishburne Military Institute and the University of Virginia from which he obtained the bachelor of science and doctor of medicine degrees. He spent sixteen months at Lakeside Hospital, Cleveland, Ohio, and later at the Manhattan Eye, Ear and Throat Hospital in New York, he was a resident and an assistant surgeon. He was assistant otolaryngologist at St. Lukes' Hospital and clinical assistant in otolaryngology at Bellevue Hospital, also in New York.

He was a talented musician who enjoyed playing both classical and popular music. His hobby was colored photography; his religion, Catholic. Commander Chew was a member of the Medical Society of the State of New York, the American Triological Society and the American Academy of Ophthalmology and Otolaryngology.

Commander Chew volunteered for military service in June, 1942, and was on duty in Bancroft Hall at the Naval Academy, Annapolis. He died September 4, 1943, at the age of 39, leaving his wife, the former Katharine L. Salmon, and two brothers who are in the United States Army.

WILLIAM FRANKLIN CLEVENGER

Dr. Clevenger was born November 11, 1874, in Missouri, and attended school in Warrensburg, Mo. At the Indiana Medical College in Indianapolis he obtained his medical degree in 1894. Dr. Clevenger was fortunate in being able to attend European clinics for study in London, Paris and Berlin. His practice of otolaryngology began in 1898 in Indianapolis, Ind. He was on the staffs of St. Vincent's, Methodist and City hospitals.

Besides belonging to state and county medical societies, he was a Fellow of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association, and the American College of Surgeons. During World War I he served as a lieutenant colonel in the medical reserve corps, U. S. Army.

Dr. Clevenger died November 20, 1943, in Indianapolis. He is survived by his wife and 2 daughters.

JOHN HAMILTON COOPER

Dr. Cooper was born in 1870 in Milledgeville, Pa. He studied at McElwain Institute, Washington and Jefferson College and received his medical degree from the University of Pittsburgh School of Medicine in 1913. He was a pharmacist as well. The University of Vienna and the Royal Hungarian Hospital conferred degrees on Dr. Cooper for postgraduate study.

In 1915, he started practice in Brewster, Ohio, but two years later moved to Massillon, Ohio. Dr. Cooper was on the staffs of the Massillon City Hospital, and the Massillon State Hospital. Besides belonging to local and state medical societies, he was a Fellow in the American Academy of Ophthalmology and Otolaryngology, having been elected in 1940.

Dr. Cooper was affiliated with the Presbyterian church. He was also a thirty-second degree Mason. Death occurred July 15, 1943, in Massillon. He is survived by two brothers and a sister.

WILLIAM THORNWALL DAVIS

Born in Little Rock, Ark., January 14, 1877, Dr. Davis attended the Kentucky Military Institute before going into college work. In 1901 he received his doctor of medicine degree from the George Washington University School of Medicine. The next year was spent at the U. S. Army Medical School and further postgraduate work as taken at Vienna in 1906.

In 1902 Dr. Davis began his service with the medical corps of the U. S. Army. As lieutenant and later captain under General Leonard Wood, he took part in the Moro campaign in the Philippines in 1904-05. He was also in the Datu Ali campaign under General Frank McCoy in 1905. In World War I he was a major in the medical corps.

Settling in Washington, D. C., Dr. Davis became professor of ophthalmology at the U. S. Army Medical School in 1917-18. From 1920 on he held a similar teaching appointment at the George Washington University School of Medicine. In addition to being senior surgeon at the Episcopal Eye, Ear, Nose and Throat Hospital, he was also consulting ophthalmologist at Garfield, Columbia,

Gallinger, Casualty and George Washington Hospitals. From February, 1943, to May, 1944, he served the Surgeon General as a consultant in ophthalmology. Dr. Davis was a prolific writer of scientific papers and made many contributions to his field.

A Fellow of the American Academy of Ophthalmology and Otolaryngology, of which he was first vice president in 1935, the American College of Surgeons, the American Medical Association, and the Southern Medical Association, he also belonged to the Medical Society of the District of Columbia and the Washington, D. C., Ophthalmological Society. He was a member of several military orders, of Phi Sigma Kappa and of Sigma Xi. He was a Mason, a Rotarian, and belonged to the Episcopal church.

His wife, Mrs. Renee Davis, and sons, Dr. William J. G. Davis, Major Roger Davis, Lt. Renee Sheldon Davis and Akin Thornwall Davis survive. Death occurred June 16, 1944, in Washington, D. C.

LEE WALLACE DEAN

Lee Wallace Dean, professor emeritus of otolaryngology, Washington University School of Medicine, St. Louis, and a former President of the American Academy of Ophthalmology and Otolaryngology, passed away February 9, 1944.

Born in Muscatine, Iowa, March 28, 1873, Dr. Dean studied at the University of Iowa and received his degree of doctor of medicine there in 1896. After studying in Vienna a year he returned to Iowa to practice medicine. At the State University of Iowa, Dr. Dean served as professor and head of the department of otolaryngology and oral surgeon from 1900 to 1927. From 1912 to 1927 he also served as dean of the medical school. He then went to St. Louis as professor of otolaryngology of Washington University School of Medicine and subsequently he became director in research in otolaryngology at the Oscar Johnson Research Institute.

Besides being otolaryngologist-in-chief at the McMillan Eye, Ear and Throat Hospital, he was a member of the staff of that institution and also of Barnes Hospital, St. Louis Children's Hospital, Jewish Hospital and St. Louis Maternity Hospital. He was otolaryngologist-in-chief to outpatients at the University Clinics.

Dr. Dean was a Life Fellow in the American Academy of Ophthalmology and Otolaryngology, having been elected to membership in 1898. He served as President in 1937. He was also at one time president of the Iowa State Medical Society, American Laryngological Association, American Laryngological, Rhinological and Otological Society, American Otological Society and secretary of the Section on Laryngology, Otology and Rhinology of the American Medical Association. Dr. Dean was a member of other scientific groups including the Association of American Peroral Endoscopists, Missouri State Medical Association, American Broncho-Esophagological Association, and La Societe de laryngologie des hopitaux de Paris.

During World War I, he was commanding officer of General Hospital No. 54 and was a lieutenant colonel in the medical officers reserve corps, retired. In 1937, he was honored with the de Roaldes Gold Medal of the American Laryngological Association and from 1927 until the time of his death, Dr. Dean was editor-in-chief of the Annals of Otology, Rhinology and Laryngology.

ZENAS HORACE ELLIS

Dr. Ellis was born February 14, 1895, in Poultney, Vt. After attending public school and the Troy Academy, he entered the University of Vermont from which he graduated with an A.B. degree in 1917. His M.D. degree was obtained in 1920 from the University's College of Medicine. Later he worked at Bellevue and Mary Fletcher Hospitals.

In 1924 Dr. Ellis began practice with Dr. John Wheeler and Dr. Charles May in New York. At one time he was an instructor in ophthalmology at the New York University College of Medicine, and also an assistant professor of anatomy at his alma mater.

Dr. Ellis was a Fellow of the American Academy of Ophthalmology and Otolaryngology and the American Medical Association. He also belonged to the Medical Society of the State of New York and to other local societies.

He attended the Presbyterian Church and also held a membership in the University Club of New York City. Date of death was October 20, 1943. His wife, Mrs. Mignonette Peterson Ellis, and his mother, Mrs. Edward E. Ellis, survive him.

ROBERT JOHN FERGUSON

Dr. Ferguson was born September 11, 1871, in New York City. After attending public schools, Rice Academy and Spring Garden Institute, he graduated from Hahnemann Medical College, Philadelphia, in 1899. Settling in New Haven, Conn., Dr. Ferguson was connected with Grace Hospital for many years. Postgraduate work was taken at the Philadelphia Polyclinic, New York Ophthalmic Institute, Mayo Clinic, Rochester, Minn., and in Berlin and Vienna.

In addition to state and local medical societies, he was a fellow of the American Medical Association and the American College of Surgeons. He was certificated by the American Board of Otolaryngology and elected to Fellowship in the American Academy of Ophthalmology and Otolaryngology in 1927.

Dr. Ferguson was an ardent traveler in both the United States and Europe. His hobbies were golf and fishing. He erected the first Professional building in Connecticut. He was affiliated with the Cheshire Congregational Church and was a member of the New Haven Commandery and Rotary Club.

He died May 15, 1944, in New York City, and is survived by two daughters, Mrs. R. Blake Russell, Mrs. A. Bryan Clark, Jr., and five grandchildren.

CARL FISHER

The loss of Dr. Fisher, June 7, 1944, in Los Angeles, Calif., is one that is keenly felt in Los Angeles. His place will be hard to fill. Aside from his medical interests, he was civic-minded and sponsored many worth while projects in art and music in the community. He was in constant demand as a toastmaster at both medical and social gatherings because of his cleverness in impromptu speaking and his ready wit.

Born July 16, 1879, in Breckenridge, Minnesota, Dr. Fisher attended public schools, then Carleton College, Northfield, Minn., where he won his A. B. degree in 1901. In 1905 he graduated from Harvard Medical School. Internship was served at the Massachusetts Charitable Eye and Ear Infirmary to 1907, and visits made to various European clinics.

In 1909 Dr. Fisher accepted the position of head of the section on ophthalmology and otology at the Mayo Clinic, Rochester, Minn. He resigned in 1917 to become a captain, then major in the medical corps of the A.E.F. and served overseas in several Base Hospitals during World War I.

In 1920 he went to Los Angeles to practice. He was associated with the Good Hope Clinic, Good Samaritan, St. Vincent's and Los Angeles Eye & Ear Hospitals. He was also associate professor of ophthalmology at the University of Southern California School of Medicine.

The American Board of Ophthalmology certificated Dr. Fisher in 1920, after which he became a Fellow of the American Academy of Ophthalmology and Otolaryngology. Other society affiliations were with the American Medical Association, American College of Surgeons, American Ophthalmological Society, Los Angeles Ophthalmological Society and the Los Angeles Clinical and Pathological Society. Dr. Fisher was one of the original committee of the Research Study Club of Los Angeles.

GEORGE FOSTER FISKE

Dr. Fiske was born in Madison, Conn., January 26, 1860. At the age of 16 he entered Amherst College where he excelled scholastically, being awarded the Sawyer prize for the best examination in anatomy and physiology in his sophomore year as well as a $50.00 gold medal for examination on the ear. He was elected to Phi Beta Kappa his junior year and graduated in 1881. Before entering Yale Medical school, Dr. Fiske worked in St. Louis for a time. By intensive study and because of his special work at Amherst, Dr. Fiske was able to combine several years of medical school and he received his M.D. degree with the class of 1883. Upon graduation he sailed for England, toured the country on bicycle, and then traveled to Germany where he visited many university towns. In Halle, he met Professor Alfred Grafe who said he could work in the clinic there. Dr. Fiske remained at Halle for nearly two years working with Professor Grafe and also with Professor Schwartze in his ear clinic.

Before returning to the States in 1885, Dr. Fiske visited other clinics in Germany, Austria, Switzerland and France. In January, 1886, he began practice in Chicago with Dr. Truman Miller. Later he became Professor of Otology in the Chicago Polyclinic, surgeon at St. Lukes Hospital, and a founder of the Henrotin Hospital. He was a fellow of the Ameri-

can Medical Association, the American Otological Society, Inc., the American College of Surgeons and the Chicago Ophthalmological Society of which he was three times president. In 1903, he was elected to Fellowship in the American Academy of Ophthalmology and Otolaryngology.

As a young man Dr. Fiske was very active athletically. He was a noted bicyclist and mountain climber. He played golf and tennis winning many trophies. He was also a contract bridge enthusiast and wrote a little book on the subject that became very popular.

Dr. Fiske's death occurred October 18, 1943, in Chicago. He is survived by his widow, Edna Withers Fiske, and two sons by his first marriage, Samuel Fiske and Dr. George F. Fiske, Jr.

ARTHUR EMILE GADBOIS

Dr. Gadbois was born November 30, 1874, in Sioux City, Iowa. He attended Nicollet College, Quebec, Canada, and received his medical degree from Creighton Medical College, Omaha, Nebr., in 1903. He began his practice that year in Rogers, Nebr., and later moved to Norfolk in the same state, where he was otolaryngologist and ophthalmologist to the Lutheran Hospital and Our Lady of Lourdes Hospital. He was also division ophthalmologist for the Chicago and Northwestern Railway.

Dr. Gadbois took postgraduate work at the Chicago Eye and Ear Infirmary, New York Polyclinic and in Vienna at the Allegemeinde Krankenhaus. He was a diplomate of the American Board of Otolaryngology and became a Fellow of American Academy of Ophthalmology and Otolaryngology in 1921. He was also associated with the Nebraska State Medical Society and the local county association. He was a member of the Roman Catholic Church.

Dr. Gadbois' wife, three sons and one daughter survive him. He died November 19, 1943, in Norfolk.

SANFORD ROBINSON GIFFORD

Sanford R. Gifford who died in Chicago on February 25, 1944, was born in Omaha, Nebr., January 8, 1892. Following his preliminary education in his home town, he entered Cornell where he majored in literature, a taste that he maintained to the end. His M.A. and M.D. degrees were given by the Universiy of Nebraska, the latter in 1918. After a short internship in the Wise Memorial Hospital, he was assigned to Base Hospital 49 as bacteriologist in France and later in the Army of Occupation. Subsequent to discharge, a two years' internship was served in the Nebraska Methodist Hospital. Another two years were spent in the University Ophthalmic Clinic and in practice with his father, the distinguished Dr. Harold Gifford. This was followed by a European study tour with much of the time spent in Freiburg with Dr. Axenfeld and in Paris with Dr. Morax. He then returned to practice with his father in Omaha until 1929 when he was called to the Chair of Ophthalmology at Northwestern University in Chicago. In February of 1944, he contracted the virus pneumonia that proved fatal. He is survived by his wife and two children.

It is difficult to speak to Sandy Gifford in terms of moderation, for everything that he did, he did superlatively well. His ophthalmic achievements are too well known to Fellows of the Academy to necessitate enumeration here as was his enthusiasm in scientific matters. That same enthusiastic outlook dominated his social as well as his intellectual life and made him a most refreshing companion, either in private concourse or on the floor of a scientific meeting. The fauna of the South Sea Islands, the characteristics of active volcanoes, the habits of migratory birds, the behavior of peripheral arterioles, were all grist to his mill, provided they were presented in a scientific manner.

Sandy had that rare ability of attracting earnestly minded young men to his side with the resultant development of a "school." He and his associates were prolific writers and contributed much in books and special articles. By these writings, by his ability, and by his personality, Sandy Gifford created an indestructible niche for himself in the Hall of Fame of American Ophthalmology.

HERBERT MASKELL GODDARD

Dr. Goddard was born July 26, 1880, in Boston, Mass. His early education was obtained in Boston, Nashotah, Wis., and at St. Stevens in Annadale ,N. Y. In 1905 he received his doctor's degree from the Medico-Chirurgical College of Philadelphia. Postgraduate work was done at the Polyclinic Hospital of Philadelphia and in 1907 he began his practice of medicine in that city. He was chief of otolaryngology at the Jewish Hospital and at Phila-

delphia General. He was also associated with the Stomach Hospital, the Jewish Orphanage, School Lane, Pa.; was a consultant of the State Hospital at Nanticoke, Pa., and assistant professor in the department of otolaryngology at the University of Pennsylvania Graduate School.

A fellow of the American Medical Association, the American College of Surgeons and a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, Dr. Goddard was also a member of the Philadelphia Laryngological Society, the Philadelphia County Medical Society, and other state and local associations. For two years preceding his death he was Coroner of the City of Philadelphia, and for the past twenty years he had been active on the Board of Trustees of the Eastern State Penitentiary. He was a devoted public official and humanitarian.

Dr. Goddard passed away November 23, 1943. He is survived by his wife, Janet Waddell Goddard, and two sisters, Mrs. J. F. Wagner of New York City and Mrs. Dickson Smith of Wyland, Mass.

GEORGE ROBERT GOWEN

Dr. Gowen was born February 20, 1882, in Bedford, Ind. His preliminary education was secured in Bedford and Terre Haute, and in 1909, he was graduated from the Southwestern Homeopathic Medical College and Hospital, Louisville, Ky. In October of 1910, Dr. Gowen started a general practice in Milton, Ore. He took postgraduate work at Harvard and at the Chicago Eye and Ear Infirmary and in 1918 he began specializing in eye, ear, nose and throat work in Walla Walla, Wash. He was a member of the Washington State Medical Society, the Pacific Coast Oto-Ophthalmological Society, past president of the Walla Walla Valley Medical Society and a Fellow of the American Academy of Ophthalmology and Otolaryngology. He was on the staffs of the Walla Walla Sanitarium and Hospital and St. Mary's Hospital. He was a member of the local Rotary club and the Masonic lodge.

Dr. Gowen died September 2, 1943, in Walla Walla. He is survived by his wife, a daughter and two sisters.

JOHN MILTON GRISCOM

Dr. Griscom was born February 23, 1881, in Salem, N. J. He attended Swarthmore College and received his doctor's degree from the University of Pennsylvania. In 1908 he began his practice of medicine in Philadelphia.

Dr. Griscom was attending surgeon at Presbyterian and Methodist Hospitals, executive surgeon at Wills Eye Hospital, and professor emeritus of ophthalmology at the University of Pennsylvania Graduate School. He was consulting ophthalmologist at Jeanes Hospital, Philadelphia, and at Burlington County Hospital, Mt. Holly, N. J.

His church affiliation was with the Society of Friends. He was a fellow of the American Medical Association, a member of state and county societies, the College of Physicians of Philadelphia, and the American Ophthalmological Society of which he was editor, secretary and president of the council at different times. He also belonged to the American Academy of Political and Social Science. Dr. Griscom was elected to Fellowship in the American Academy of Ophthalmology and Otolaryngology in 1920.

Dr. Griscom died June 5, 1943. Surviving are his wife, Mary Lippincott Griscom, and a daughter, Mary Griscom Colegrove.

ISRAEL GRUSHLAW

Dr. Grushlaw was born September 15, 1876, in Russia. He attended school in Berlin and New York City and obtained his doctor's degree at the University of Pennsylvania School of Medicine in 1899, after which he began practice in New York City. His practice was limited to diseases of the ear, nose and throat.

Dr. Grushlaw entered the otolaryngological service of Lebanon Hospital in 1910 and in 1922 he was appointed Associate Attending. He was also connected with Peoples Hospital, Gouverneur Hospital and the Manhattan Eye, Ear and Throat Hospital. He was a founder and one time president of the New York Physicians Association. A member of the New York County Medical and the Eastern Medical Society, he became a Fellow of the American Academy of Ophthalmology and Otolaryngology in 1905.

Dr. Grushlaw was a well-known book and art collector. His art library was one of the finest in the country. He was also the author of several scientific papers and he designed his own instruments for use in tonsillectomies.

His death occurred December 9, 1943, in New York City. Surviving are his wid-

ow, Mrs. Isabelle F. Grushlaw, and two daughters, one of whom is an artist, the other a psychologist.

WILLIAM CARLETON HARRIS

Dr. Harris was born November 30, 1870, in Covington, Ky. Following his graduation from the College of Pharmacy of Cincinnati he began the study of medicine and in 1897, was graduated from Miami Medical College in Cincinnati.

Dr. Harris was a member of the Deaconess Hospital staff for forty-five years. He was the first resident physician of the Jewish Hospital and retained his membership on that staff through the years that followed. He was an Episcopalian by religion, a member of the Masonic Order, the Elks, and a founder of the Otolaryngological Society of Cincinnati. Dr. Harris was elected to the American Academy of Ophthalmology and Otolaryngology in 1900 and was elevated to Life Fellowship in 1935. His other society affilations included the Ohio State Medical Association, American Medical Association and the Cincinnati Academy of Medicine.

Dr. Harris played a prominent role in the affairs of the General Protestant Orphans' Home, where, for many years, he rendered free medical service to the wards, and gave freely of his time to charitable projects of that institution.

Dr. Harris died at the Deaconess Hospital, August 12, 1943. Surviving him are his wife, Blanche Thomas Harris, and a daughter, Mrs. Allan Walsh.

RAYMOND WILLARD HAWKINS

Born April 4, 1894, at Gainsville, N. Y., Dr. Hawkins prepared for his career at the University of Rochester where he obtained his B. S. degree in 1916. His medical training was received at Johns Hopkins University School of Medicine, Baltimore, from which he graduated in 1920.

Internship followed at the Long Island College Hospital, Brooklyn, and residency at St. Luke's Hospital, Chicago, in the eye, ear, nose and throat service. In 1925, Dr. Hawkins began practice in Rochester, N. Y. He was assistant otolaryngologist at Strong Memorial Hospital, attending otorhinolaryngologist at Rochester General and Highland Hospitals, and since 1925 an instructor in otorhinolaryngology at the University of Rochester Medical School.

Dr. Hawkins was a fellow of the American College of Surgeons, American Medical Association, American Academy of Ophthalmology and Otolaryngology, and a member of the Rochester Pathological Society as well as other local medical societies.

Dr. Hawkins was fond of local history and country life. He passed away March 30, 1944, at Conesus, N. Y., and is survived by his wife Anita B. Hawkins and a daughter Mary Patricia.

ERNEST REED HIRST

Dr. Hirst was born in Camden, N. J., June 6, 1891. Educated in the Camden public schools and high school, he went to the Jefferson Medical College of Philadelphia and obtained his doctor of medicine degree there in 1918. Upon graduation he returned to his home town to take up the practice of ear, nose and throat work. In 1921-22, he did postgraduate work at the University of Pennsylvania and in 1924-25, he studied bronchoscopy under Dr. Chevalier Jackson.

Dr. Hirst was a member of the Camden County Medical Association, the New Jersey Medical Association, and a fellow of the American Medical Association. Certificated by the American Board of Otolaryngology, he became a Fellow of the American Academy of Ophthalmology and Otolaryngology in 1937. He was on the staff of the Cooper Hospital, Camden, N. J., and the Zurburg Memorial Hospital in Riverside, N. J. He was a member of St. Paul's Protestant Episcopal Church.

Dr. Hirst passed away January 27, 1944, leaving his wife, a daughter and a son.

SAMUEL IGLAUER

On June 23, 1944, Cincinnati lost one of her most prominent medical men in the death of Dr. Samuel Iglauer. A specialist in the ear, nose and throat field, he was an expert with the bronchoscope and during the last 20 years he had become known for his skill in plastic surgery. Dr. Iglauer was associated with the Cincinnati General, Jewish and Children's Hospitals, in all of which he was director of otolaryngology. He was professor of otolaryngology at the University of Cincinnati College of Medicine for many years.

Born December 28, 1871, in Cincinnati, he attended Woodward High School, obtained his B.S. degree from the University of Cincinnati, and in 1878 graduated from the Medical College of Ohio. In

1900 he studied plastic surgery in Vienna.

Dr. Iglauer had numerous activities. Aside from many state and local societies, he was an honorary member of the Kansas City Society of Ophthalmology and Otolaryngology, the Omaha Mid-West Clinical Society, and the Ohio Public Health Association.

In the American Medical Association he served as chairman of the Ear, Nose and Throat Section in 1925-26; he was vice president and council member in the American Laryngological, Rhinological and Otological Society, president in 1923 of the American Broncho-Esophagological Association and Second Vice President in 1935 of the American Academy of Ophthalmology and Otolaryngology. He also belonged to the American Laryngological Association, the American College of Surgeons, and the University of Cincinnati chapter of Sigma Xi. During World War I he was a captain in the medical corps.

Dr. Iglauer is survived by his wife, Mrs. Helen Iglauer, two daughters, Dr. Helen I. Glueck and Mrs. Josephine Joseph, and two grandsons.

HARRY MORGAN IVINS

Dr. Ivins was born July 24, 1878, in Holland, Iowa. His B.S. degree was earned at the University of Iowa, and his M.D. degree at the State University of Iowa College of Medicine in 1908. The following year he was clinical assistant in the department of ophthalmology, otology and rhino-laryngology in the University Hospital. In 1910 he opened his practice in Cedar Rapids, Iowa, where he served on the staffs of Mercy and St. Luke's Methodist Hospitals.

Dr. Ivins was a Life Fellow in the American Academy of Ophthalmology and Otolaryngology. He also belonged to the Iowa State Medical Society, Sigma Xi and to the Elks. He was a member of the Episcopal church. Hunting, fishing and gardening were his hobbies.

Death occurred March 20, 1944, in Santa Cruz, California. He is survived by a daughter, Audrey Lea Ivins Nigh.

GEORGE ERNEST JOHNSON

Born in 1882, Dr. Johnson graduated from the Medico-Chirurgical College of Philadelphia in 1904. Later he was assistant professor of laryngology at the Medico-Chirurgical College, Graduate School of Medicine, University of Pennsylvania.

Certificated by the American Board of Otolaryngology, he was elected to Fellowship in the American Academy of Ophthalmology and Otolaryngology in 1939. He was also a fellow of the American College of Surgeons and of the American Medical Association. Dr. Johnson was chief of the division of communicable diseases for the city health department from 1934 to 1942. He served on the staff of St. Agnes Hospital and was on the visiting staff of Philadelphia Hospital for Contagious Diseases. Death occurred August 12, 1943.

GEORGE LINDSAY JOHNSON

Dr. George Lindsay Johnson, an Honorary Fellow of the American Academy of Ophthalmology and Otolaryngology, was born near Manchester, England, July 10, 1853. He was educated at Amersham Hall, near Reading, England; the University of Bonn, Kornthal in Wurttemberg, Germany; Owens College, Manchester, England and Caius College, Cambridge. Since 1900, he had practiced ophthalmology in Durban, Natal, South Africa.

Dr. Johnson made a special study of optics and did a vast amount of research in that science. He is probably best known for his work on mammalian eyes and those of reptiles and amphibia. For these works he was made an honorary fellow of the Friedrich Wilhelm Academy of Scientific Research, Berlin, and honorary fellow of the Royal Society of Italy. Well-known in the photographic world for his work in connection with the construction of lenses and color photography, Dr. Johnson was elected a fellow of the Royal Photographic Society and obtained the gold medal at the St. Louis exhibition, as well as the bronze medal. He was for ten years an examiner in theoretic and applied optics to the S. M. C., of London, and ophthalmic surgeon to the Western General Hospital Dispensary, which appointment he held for twenty years. He was also ophthalmic surgeon to the West End Hospital for Nervous Diseases and to various charities.

Dr. Johnson died in South Africa in July, 1943.

WILSON JOHNSTON

Dr. Johnston was born October 23, 1867, in Toronto, Canada. After attending public schools he studied at Collegiate Institute in Toronto, the University of Louisville and Kentucky School of Medicine where he earned his degree in medicine in 1892. He first opened a practice in Webster Springs, W. Va. In 1896, Dr. Johnston went to New York for postgraduate study in diseases of the eye, ear, nose and throat. Following this, he practiced his specialities in Moscow, Idaho. Here he met and married Miss Florence Corbett of Tacoma, Washington.

In 1917 Dr. Johnston moved to Portland, Oregon, where he has been a prominent physician for the last twenty-five years. He was associate clinical professor in the department of ophthalmology at the University of Oregon Medical School and a member of the staff of the Coffey Memorial Hospital. He was a fellow of the American Medical Association, the American College of Surgeons, a past president of both the Oregon and Washington state medical societies, a member of the Pacific Coast Oto-Ophthalmological Society, the Portland Academy of Oto-Ophthalmology, and a Senior Fellow in the American Academy of Ophthalmology and Otolaryngology.

Dr. Johnston took an active part in civic affairs; he was a vestryman of the Trinity Episcopal Church, a Rotarian, a Mason, a member of the Portland Chamber of Commerce, the Men's Garden Club of Portland, and the Multnomah Community Club. In 1942, he was appointed the Oregon state chairman of procurement and assignment service for physicians.

Dr. Johnston's death occurred August 15, 1943. His wife, a daughter and a son, Major J. Wilson Johnston, survive him.

CHARLES CURTIS JONES

Dr. Jones was born in Independence, Ky., on February 13, 1887. His early schooling was obtained in Independence and Walton, Ky. In 1910 he received his M.D. degree from the University of Cincinnati College of Medicine and in 1913 he began his practice in that city.

Dr. Jones served on the staffs of the Christ Hospital, Children's Hospital and the Bethesda Hospital and was at one time assistant professor of otolaryngology at the University of Cincinnati College of Medicine.

A diplomate of the American Board of Otolaryngology, he became a Fellow of the American Academy of Ophthalmology and Otolaryngology in 1925. He was a member of the American Laryngological, Rhinological and Otological Society, the Cincinnati Academy of Medicine, the Ohio State Medical Society, and a fellow of the American Medical Association and the American College of Surgeons. Locally he belonged to the Masonic Lodge, the Norwood Club, and was an active member of the Grace M. E. Church. Dr. Jones was also the owner of the Pine Hill Farms, Sharonville, Ohio.

He passed away November 22, 1943. Surviving are his wife, Mrs. Kathryn H. Jones; two sons, Charles R. and William F.; and two daughters, Mrs. Nell Alexander and Virginia Jones.

CARL KOLLER

With the passing of Carl Koller the medical profession lost one of its great scientists and ophthalmologists. His best known accomplishment was his discovery in 1884 of the use of cocaine as a local anesthetic in operations on the eye. Marking the fiftieth anniversary of this occasion, he was awarded the Medal of Honor of the American Academy of Ophthalmology and Otolaryngology in 1934, and at the same time was made an Honorary Fellow of the Academy.

Born in 1857 Dr. Koller studied at the Medizinische Fakultat der Universitat Wien, Austria, graduating in 1882. While there he also worked in embryology in Stricker's laboratory and published his observations on the origin of mesoderm in the chick embryo, which later proved to be correct. Dr. Koller's keen scientific mind and interest in experimentation were responsible for many other contributions, among them his design of the lighting system used in the present electric ophthalmoscope.

Many honors were conferred upon Dr. Koller. In 1922 he received a gold medal from the American Ophthalmological Society, in 1929 the Kussmaul medal from the University of Heidelberg, Germany, and in 1930 the first annual medal of honor from the New York Academy of Medicine.

In 1888 Dr. Koller started a practice in New York and was consulting ophthalmic surgeon at Mount Sinai Hospital for many years. He was also consultant to

Montefiore Hospital and the Hebrew Orphan Asylum.

His society affiliations were many and included the American Ophthalmological Society, the Association for Research in Ophthalmology, Inc., the American Physiological Society, the American Society for Pharmacology and Experimental Therapeutics, Inc., the American Medical Association and the New York Academy of Medicine. He was an honorary member of the Society of Physicians of Vienna, the Society of Physicians of Budapest and the Royal Academy of Medicine, Rome, Italy.

Dr. Koller died March 22, 1944.

WILLIAM ANDREW KRIEGER

Dr. W. A. Krieger, a vice president of the American Academy of Ophthalmology and Otolaryngology in 1943 and an eminent eye, ear, nose and throat specialist of Poughkeepsie, N. Y., was born March 16, 1883, in Poughkeepsie. Educated in the local schools, he received his medical degree from Albany Medical College, Albany, N. Y., in 1906. The next year he served his internship at Albany City Hospital and the following year he spent in the Bender Laboratory of the same city. From 1908 to 1910, Dr. Krieger was in general practice in Poughkeepsie. He then became associated with Dr. W. G. Dobson, an ophthalmologist and otolaryngologist, until 1918, at which time he enlisted in the Army Medical Corps and served until April, 1919.

Dr. Krieger made several trips abroad studying in Paris, Vienna and London. He also studied with Dr. H. P. Mosher in 1920. He was consulting ophthalmologist and otolaryngologist at Vassar Brothers Hospital, having been associated with that institution for twenty-nine years. He was also consultant at Hudson River State Hospital and Bowne Memorial Hospital in Poughkeepsie. In addition, he served Harlem Valley State Hospital, Wingdale, N. Y.; Matteawan State Hospital, Beacon, N. Y.; Butterworth Hospital, Cold Spring, N. Y.; Wassaic State School, Wassaic, N. Y.; and Sharon Hospital, Sharon, Conn.

He was a past president of the Dutchess County Medical Society. He was secretary, then chairman of the eye, ear, nose and throat section of the New York State Medical Society and for ten years was chairman of Scientific Exhibits of that state society. In April, 1941, he was elected first vice president of the New York Society for the ensuing year. A fellow of the American Medical Association, the American College of Surgeons, the New York Academy of Medicine, he also belonged to the Pan-American Medical Association. In 1919, he was elected to Fellowship in the American Academy of Ophthalmology and Otolaryngology. He was a life member of the Poughkeepsie lodge, Free and Accepted Masons. He attended Christ Episcopal Church. Dr. Krieger had been president of the Merchants' National Bank and Trust Company since 1934.

His death occurred suddenly at his home, May 19, 1944. His wife, Bessie Livingston Krieger, whom he married in 1923, survives him. In his death, the profession and community at large suffered a loss that it could ill afford to sustain.

MAX LUBMAN

Dr. Lubman was born in Russia in 1876. He attended Cornell University Medical College, New York, and graduated in 1900. A year later he began practice in New York City.

He was connected with the Har Moriah Hospital in 1901. From 1910 to 1928 he served at Mt. Sinai Hospital in the ear, nose and throat out-patient department. A Fellow of the American Medical Association and the American Academy of Ophthalmology and Otolaryngology, Dr. Lubman also was affiliated with local medical societies.

Music composition was one of his outside interests. Death occurred November 20, 1943. Surviving are his wife and a daughter.

CHARLES HENRY MAY

Dr. May, son of Henry May and Henrietta Oppenheimer May, was born in Baltimore, August 7, 1861. Four years later his family moved to New York City, his future home. He was a brilliant student and was graduated from the College of Pharmacy of the City of New York in 1879 with the award of a gold medal. In 1883 he received his degree of doctor of medicine from the College of Physicians and Surgeons, Columbia University. Here also he received many scholastic honors which led to his nomination to the position of Junior Assistant to Bellevue Hospital; however, he had previously accepted on appointment at Mt. Sinai Hospital and there remained for two years. Dr. May entered private practice in 1885,

and partly through his friendship with Cornelius R. Agnew, professor of ophthalmology at the College of Physicians and Surgeons, he decided to enter the speciality of diseases of the eye. In 1887, he went to Europe for postgraduate study, spending six months at Halle, a month in Berlin, six months in Vienna, and a few weeks in London and Paris.

On returning to New York he received appointments at the Manhattan Eye and Ear Infirmary, the New York Ophthalmic and Aural Institute, and the College of Physicians and Surgeons. He also became associated with Vanderbilt Clinic, Polyclinic and Mt. Sinai Hospital. In 1914, he established the ophthalmologic service at Bellevue Hospital where he remained as director until 1925.

Dr. May's private practice was very large and medical visitors from all over the world visited his office. In 1900 he devised an improved double disc ophthalmoscope and in 1914 he introduced the improved system of illumination for electric ophthalmoscopes. He was a prolific writer, his most widely known work being his MANUAL OF DISEASES OF THE EYE which has gone through eighteen editions since 1900 and been translated into ten foreign languages. He was an artist of ability in painting fundus lesions, a skillful surgeon, and a careful diagnostician whose ethical standards were ideal.

He was a member of the American Ophthalmological Society, the New York Academy of Medicine, the American College of Surgeons, the American Medical Association and New York state and county medical societies. He was a Life Fellow of the American Academy of Ophthalmology and Otolaryngology having been elected to the Academy in 1904.

His death occurred December 7, 1943. Surviving are his wife and two sisters.

MYRON FIRTH METZENBAUM

Dr. Metzenbaum was born on April 1, 1876, in Cleveland, Ohio. He became a life long resident of this city. In 1900 he received the degree of doctor of medicine from the University of Wooster Medical Department and in 1901 he began his practice of medicine in Cleveland. From 1902-1905, he lectured on anatomy at Western Reserve University. He served on the staffs of the Huron Road Hospital, East Cleveland, Mt. Sinai and St. Lukes' Hospitals. Dr. Metzenbaum made several trips to Europe where he studied in Vienna, Paris, London and Berlin. He was certificated by the American Board of Otolaryngology and was a member of the founders group of the American Board of Plastic Surgery.

A Life Fellow of the American Academy of Ophthalmology and Otolaryngology which he joined in 1908, Dr. Metzenbaum was also a fellow of the American College of Surgeons and the American Medical Association and a member of the Cleveland Otolaryngological Club, the Cleveland Academy of Medicine and the European Congress of Reconstructive Surgery. In 1904, at the St. Louis Exposition, he was awarded a government medal for research in radium.

In 1900, Dr. Metzenbaum developed and introduced the method of administering ether-air or drop ether anesthesia. In 1909, he established Cleveland's present ambulance system under the police department. This system has since been adopted throughout the country. He was the author of numerous papers on radium, anesthesia, and nose, throat and larynx surgery. Dr. Metzenbaum attended the Ansel Rd. Temple and was a member of the Oakwood Golf Club. He died January 25, 1944, leaving a widow, Elsa F. Metzenbaum, and two daughters.

JOSEPH EDWARD MILLER

Dr. Miller was born June 6, 1889, in Elvaston, Ill. His early education was obtained in Keokuk, Iowa, and in 1912, he was graduated from the Wisconsin College of Physicians and Surgeons in Milwaukee. In 1913 Dr. Miller began practicing medicine in Wayland, Mo. Later he became established in Los Angeles, Calif.

In 1927 Dr. Miller began limiting his practice to eye, ear, nose and throat work. He did postgraduate work at Rush Medical College in 1927-28 and in 1934, was certificated by the American Board of Otolaryngology.

Dr. Miller was a member of the Los Angeles County Medical Society, the California State Medical Association, the Pacific Coast Oto-Ophthalmological Society, the Los Angeles Society of Ophthalmology and Otolaryngology, and he was a fellow of the American Medical Association and the American College of Surgeons. In 1940 he became a Fellow of the American Academy of Ophthalmology and Otolaryngology.

Dr. Miller was on the staff of the Marian Davies Clinic and the White Memorial Clinic in Los Angeles. He was also on the staff of the Santa Monica Hospital and St. John's Hospital in Santa Monica. He was a member of the Lutheran church.

Dr. Miller died September 13, 1943, in Los Angeles. He is survived by his wife, three sons who are all in the armed forces, and one daughter.

JOHN W. MILLETTE

Dr. Millette was born on June 16, 1864. He studied in Ohio, attending Ohio Northern University, Ohio Wesleyan University and Wittenburg College where he won his Master of Arts degree. He studied medicine at Ohio Medical University, graduating in 1903. It is interesting to note that before going into the medical profession, Dr. Millette taught school for several years. His last position was that of principal of the public school at New Carlisle, Ohio.

Before opening his office in Dayton, Ohio, in 1905, he visited medical centers in New York, London and Vienna. Dr. Millette was on the staffs of the Veterans Administration Facility, National Military Home and St. Elizabeth Hospital for many years.

Dr. Millette belonged to the Masonic Order and was a Shriner. Professional organizations in which he participated include the American Academy of Ophthalmology and Otolaryngology, American Medical Association, American Association for the Advancement of Science, Ohio State Medical Society, and local medical societies.

An ardent traveler, he visited Hawaii, South America, Bermuda, Cuba, Mexico and Europe. Death occurred December 28, 1943, leaving his wife and a daughter.

MILLEN ALEXANDER NICKLE

Dr. Nickle died March 6, 1944, in Clearwater, Fla., where he had resided for 20 years. Born in Madoc, Ont., Dec. 31, 1881, he attended Albert College, Belleville, Ontario, and obtained his M.D. degree from Toronto University in 1907. Postgraduate training was taken at Herman Knapp Memorial Hospital, New York, and in other medical centers in this country and in Europe.

He began practice in 1910 in Weyburn, Sask., and was connected with the Toronto, Weyburn, and Barrie, Barrie, Ont., General Hospitals and was a surgeon for the Canadian Pacific Railroad. In World War I, Dr. Nickle was a captain in the Canadian Army Medical Corps. He was also a friend and classmate of the late Dr. Allen Dafoe, who brought the Dionne quintuplets into the world.

A fellow of the Royal College of Surgeons, the American College of Surgeons, and the American Medical Association, he was elected to Fellowship in the American Academy of Ophthalmology and Otolaryngology in 1940. He was an Elder in the Peace Memorial Presbyterian Church in Clearwater, and also belonged to the Clearwater Country Club, Yacht Club and Rotary Club. He is survived by his wife and one daughter, Mrs. H. P. Hayden of Clearwater, and two brothers and a sister who live in Canada.

WILLIAM WILSON PEARSON

Dr. Pearson was born October 21, 1869, in Illiopolis, Ill. Although he was destined to become a doctor, he was very fond of sports and played football and baseball throughout his college period. He could have been captain of either team while he was at the University of Michigan Medical School, but refused because he considered his professional work more important than athletics. However, in 1892 he was nominated for the All-American football team and was the first man west of the Alleghenies to receive this honor.

He graduated from medical school in 1893. Before opening his office in Des Moines, Iowa, in December, 1896, he had spent a year and a half in Springfield, Ill., as an assistant and the rest of the time studying abroad in Germany, Vienna, Paris and London.

Dr. Pearson was interested and very active in his society affiliations. He helped found and lead several local medical societies. He was a founder of the American College of Surgeons and a member of its Board of Governors since 1913. He was vice president in 1929. He was vice president of the American Academy of Ophthalmology and Otolaryngology in 1939 and was dean of Drake University Medical School from 1909 until it closed. He also was affiliated with the American Laryngological, Rhinological and Otological Society, and the Association for Research in Ophthalmology, Inc.

Dr. Pearson died February 11, 1944, and is survived by his wife.

REGINALD STIRLING PENTECOST

Born in Hamilton, Ontario, in 1886, Dr. Pentecost was educated in Toronto schools and graduated in medicine with honors from the University of Toronto in 1909. He took postgraduate work in New York and Vienna and visited other European medical centers, returning to Toronto in 1914.

Shortly after the outbreak of World War I he joined the Royal Canadian Army Medical Corps, going overseas in 1914 with the rank of captain. In 1917 he was promoted to the rank of Lieutenant Colonel in command of the 14th Canadian Field Ambulance. In 1918 he was consulting specialist serving with the Canadian Army in England.

After the war Dr. Pentecost began his practice in Toronto. He was assistant surgeon in the Department of Oto-Laryngology in Toronto Western Hospital, surgeon in charge of Oto-Laryngology in Christie Street Hospital, and consulting specialist to the Department of Pensions and National Health. In Christie Street Hospital he did outstanding work and made many contributions to the surgery of cancer of the larynx.

Dr. Pentecost was prominent in the Toronto Academy of Medicine, and served as president in 1932-33. He was also a Fellow of the American Academy of Ophthalmology and Otolaryngology, American College of Surgeons, Royal College of Surgeons and the Canadian Medical Association. He was a member of St. Paul's (Anglican) Church and belonged to several local social clubs.

His wife, the former Jean Isobel Henry, and their two daughters, Barbara Jean and Nancy Rex, were left after his death August 23, 1943.

ADOLPH O. PFINGST

Dr. Pfingst was born in Louisville, Ky., September 8, 1869. After graduating from high school there and securing a B.A. degree from the University of Louisville, he entered the Louisville Medical College from which he obtained his medical degree in 1891. After short services with the Louisville City and Mt. Sinai hospitals he went to Germany to study ophthalmology. In 1894 he obtained a medical degree from the University of Berlin.

Returning to this country in 1895, Dr. Pfingst became assistant to Professor Herman Knapp and house surgeon to the New York Ophthalmic Institute for a year. He then moved to Louisville, and was an instructor at the University of Louisville School of Medicine. In 1906 he was made Professor of Ophthalmology and director of the eye clinic at the Louisville City Hospital. When he retired from active teaching in 1938 the title of Professor Emeritus was conferred upon him. Dr. Pfingst's interest was centered on pathological studies of the eye and on diseases and anomalies of intracranial blood vessels.

A keen interest in professional organizations was marked by Dr. Pfingst's fellowship in the American College of Surgeons, American Ophthalmological Society, American Academy of Ophthalmology and Otolaryngology, and the American and Southern Medical Associations. He was a member of the International Society of Ophthalmology and the Pan-American Congress of Ophthalmology. State, county and local medical clubs also included him among their number. He organized the first sight saving class in Louisville and was active in national movements for the prevention of blindness.

Dr. Pfingst died February 25, 1944, leaving his wife, Mrs. Lula Salger Pfingst.

GROVER HEWITT POOS

Dr. Poos was born in Okawville, Ill., July 12, 1884. His early education was obtained there after which he attended Valparaiso University in Valparaiso, Ind. He received his doctor's degree in 1905 from the St. Louis College of Physicians and Surgeons. He did postgraduate work in Chicago and at Washington University in St. Louis.

Dr. Poos was certificated by the American Board of Ophthalmology in 1927. In 1928 he became a Fellow of the American Academy of Ophthalmology and Otolaryngology. He practiced medicine for eighteen years in St. Louis where he was clinical instructor at Washington University and on the staffs of De Paul, Barnes, and Evangelical Deaconess Home and Hospital.

Four years before his death, Dr. Poos and his family moved to Palm Springs, Calif., because of his health. He continued to practice his specialties and to take an active part in civic activities. He was a member of state and county medical societies and a fellow of the American Medical Association and the American College of Surgeons.

He died at the Coachella Valley Hospital, Indio, Calif., May 29, 1943. He is survived by his widow, Mrs. Georgiana Poos; one son, Lt. Comdr. Robert S. Poos, U.S.N. (MC); and a daughter, Mrs. Margaret Anderson.

EDWARD HYNDSHAW PORTER

Dr. Porter, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, had practiced in Tiffin, Ohio, for forty-three years. He was born in Chicago, February 28, 1873, and while still a child, moved to Detroit with his family. He attended high school there, and graduated from Beloit College, Beloit, Wis., in 1893. He was a member of Beta Theta Pi fraternity. While studying medicine at Wayne University College of Medicine, Detroit, he taught mathematics in a local high school. He received his medical degree in 1900.

Internship was served in the London Eye and Ear Hospital, London, England. Later he did postgraduate work in Vienna and Berlin, coming to Tiffin in 1901. During World War I, Dr. Porter was a captain in the medical corps. He was a fellow of the American Medical Association and was on the examining board of the American College of Surgeons. He was active in state and county and local medical societies as well as in civic affairs. He was a past president of the Tiffin Chamber of Commerce and a member of several Masonic organizations. He was affiliated with the Old Trinity Episcopal Church.

In 1902 Dr. Porter married Harriet Noble, who died five years ago. Dr. Porter died May 14, 1944, and is survived by a daughter, a son and two grandchildren. The son is serving with the Armed Forces in the Pacific theatre.

PAUL JUDD SARTAIN

Born November 26, 1861, in Philadelphia, Pa., Dr. Sartain was the son of Samuel Sartain, a well-known steel engraver, and Dr. Harriet Judd Sartain, one of the country's pioneer woman physicians. He received his B.A. degree from the University of Pennsylvania Department of Medicine in 1886. He studied later in Vienna, Berlin, Paris and London. In 1889 he started his own practice in Philadelphia.

Besides belonging to several state and local medical clubs, Dr. Sartain was a Fellow of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association, the Oxford Ophthalmological Congress, and the Societe Francaise d'Ophthalmologie. He was vice president of the Medical Club of Philadelphia in 1925.

He was also very much interested in local historical and civic affairs as is evidenced by his membership in the Franklin Institute, the Historical Society of Pennsylvania, the Zoological Society of Philadelphia, the Geographical Society of Philadelphia, of which he was secretary since 1894, and the Colonial Society of Pennsylvania. He was affiliated with the Unitarian Church and was a member of Delta Phi fraternity. His clubs were the Union League, St. Elmo and the Royal Society of London.

Death occurred April 9, 1944.

CARL FRANCIS SCHAUB

Dr. Schaub was born in Whiting, Ind., December 26, 1902. His early education was obtained in Whiting and in Prairie du Chien, Wis., and he received his A.B. degree from Marquette University in 1924. Two years later he received his B.S. degree from Loyola University and in 1928 he received an M.D. degree from that University. After a residency in Cook County Hospital, Chicago, he began the private practice of medicine in Chicago, limiting his work to diseases of the eye. He was associated with Mercy Hospital where he was senior attendant in ophthalmology and was professor and chairman of the department of ophthalmology at Loyola University School of Medicine.

Dr. Schaub was a member of city and state medical societies. He was a councilor of the Chicago Ophthalmological Society. He was elected to Fellowship in the American Academy of Ophthalmology and Otolaryngology in 1937, and was also a fellow of the American Medical Association and the American College of Surgeons. He was chairman of the Chicago group of the Patna Holy Family Hospital Association, Patna, India. Dr. Schaub was the author of several papers published in 1940 and 1941 by medical journals.

Dr. Schaub passed away January 7, 1944. His wife, Bernadette D. Schaub, and a daughter, Bernadette Mary, survive him.

FRANK L. SECOY

Dr. Secoy was born in Logan, Kan., on January 14, 1887. He went to school in

Davenport, Iowa, and in 1911 received his degree of doctor of medicine from the University of Iowa College of Medicine in Iowa City. He began practice the next year in Sioux City, Iowa, where he resided until the time of his death, January 23, 1944.

Dr. Secoy was a staff member of St. Vincent's Hospital and an associate staff member of the Methodist and Lutheran Hospitals. He was a fellow of the American Medical Association, the American College of Surgeons and a member of the Iowa Medical Association, the Woodbury County Medical Society, the Sioux Valley Eye and Ear Academy, and the Iowa State Academy of Ophthalmology and Otolaryngology. In 1922 he became a Fellow of the American Academy of Ophthalmology and Otolaryngology.

Dr. Secoy was a member of the Lutheran faith. His recreation was golfing. He is survived by a son, Frank Raymond, a brother, and a sister.

WILLIAM ALFRED SHOEMAKER

Dr. Shoemaker was born near Lock Haven, Pa., on November 10, 1859. He attended the Central State Normal School at Lock Haven and the University of Maryland School of Medicine and College of Physicians and Surgeons, Baltimore, from which he received the degree of doctor of medicine in 1885. He bagan practicing medicine that summer in Dawson, Fayette County, Pa. Postgraduate work was done at the New York Eye and Ear Infirmary during 1892-3. Following that, Dr. Shoemaker spent a year and a half in London, Heidelberg and Vienna continuing his study of ophthalmology. In 1895 he became located in St. Louis, Mo., where for the next forty years he was associated with the Washington University School of Medicine as clinical professor of ophthalmology, and at the Evangelical Deaconess Hospital as consulting ophthalmologist.

Dr. Shoemaker was elected to Fellowship in the American Academy of Ophthalmology and Otolaryngology in 1896. He was also a fellow of the American College of Surgeons, an affiliate fellow of the American Medical Association, a member of the Missouri State Medical Society and the St. Louis Ophthalmic Society. He attended St. Peter's Episcopal church in St. Louis and was a member of the Masonic Lodge. In the last ten years of his life, Dr. Shoemaker delved into many interesting subjects, preferring the arts, philosophy and sciences. He was an untiring reader. His death occurred November 7, 1943, in St. Louis. He is survived by two sisters and a brother.

RAFAEL SILVA

Dr. Silva was born in 1876 in Mexico City, Mexico. He was educated at the National Preparatory School and the School of San Ildefonso. In 1898 he received his medical degree from the University of Medicine of the City of Mexico. Internship was served at the Hospital of Jesus.

From 1904 to 1907, Dr. Silva worked in the leading clinics of Paris, Vienna, Berlin and Freiburg. In 1910, he was commissioned by the Secretary of Education and Fine Arts of Mexico to visit leading scientific centers in Europe, including clinical centers in Austria, Germany, Holland, Belgium, Switzerland, France and England. On his return in 1912, he visited clinics in the United States.

In 1916, Dr. Silva was named Professor of Ophthalmology of the National University of Mexico. In 1917 he visited ophthalmological clinics in the United States and in 1922 returned to Europe for further study. Dr. Silva served as chief of staff of the Hospital de la Luz and had many other posts in medical education and public health work. Among decorations and honors received in recognition of his contributions in these fields, Dr. Silva received the French Cross of the Legion of Honor.

Dr. Silva was affiliated with many medical organizations of Mexico, Europe, Latin America and the United States. He served as president of the Mexican Society of Ophthalmology and Otolaryngology, of the National Academy of Medicine of Mexico and of the Third Pan-American Congress of Medicine. He was a council member of the Pan-American Congress of Ophthalmology. He was treasurer and a founder of the Mexican Medical Association. He was an honorary fellow of the American College of Surgeons, a fellow of the American Medical Association and of the American Academy of Ophthalmology and Otolaryngology.

He died April 16, 1944, in Mexico City and is survived by his wife.

OTIS FRANKLIN SIMONDS

Dr. Simonds was born in Portland, Maine, April 9, 1882. He received his liter-

ary degree from Bowdoin College in 1906 and remained there for medical school from which he graduated in 1909. The next three years he interned at Boston City Hospital. Postgraduate training in ophthalmology was received at Wills Eye Hospital in Philadelphia in 1916 and in Vienna in 1921-22 at Algemeine Krandenheit.

He had practiced ophthalmology in Cleveland for 24 years before his death. He was a member of the staff of the Lutheran and St. Luke's Hospitals, the American Medical Association and the Cleveland Ophthalmological Club. He was elected to Fellowship in the American Academy of Ophthalmology and Otolaryngology in 1918 and was elevated to Senior Fellowship in 1943.

Dr. Simonds was a Delta Upsilon at Bowdoin, an active member of the Cleveland Rotary Club and also a very active member of St. Peter's Episcopal Church, participating mainly in the vestry and choir. He had traveled in Africa and at the time the Panama Canal was under construction, Dr. Simonds was situated in Ancon, Canal Zone, as a physician for the United States government.

Dr. Simonds died September 26, 1943, in Lakewood, Ohio. He is survived by his wife, Dorothy Arnold Simonds, and two sons, William Arnold and James Otis.

RAYMOND DUANE SLEIGHT

Dr. Sleight was born in Victor Township, Clinton County, Mich., in 1874. His preliminary education was obtained in Laingsburg. In 1897 he received the degree of doctor of medicine from the University of Michigan Medical School. After being in general practice for three years in Maple Rapids, Mich., Dr. Sleight went to Ann Arbor where he spent three years studying eye, ear, nose and throat work with Dr. Fleming Carrow at the University of Michigan. Postgraduate work was also done in Vienna. After his special training, Dr. Sleight settled in Battle Creek, Mich.

In World War I, he served in France in the medical corps of the United States Army. Until recently before his death he was an examiner for the local draft board and on the staffs of the Community and Leila Y. Post Montgomery Hospitals.

Dr. Sleight became a Fellow of the American Academy of Ophthalmology and Otolaryngology in 1906. He was also a member of the Association for Research in Ophthalmology, Inc., the Michigan State Medical Society and the Calhoun Medical Society.

A nature-lover, his hobbies were hunting, fishing and cultivating his flower garden.

His death occurred December 7, 1943, in Battle Creek.

LOUIS SAM SMITH

Dr. Smith was born in Brooklyn, N. Y., April 28, 1896, and attended its public schools. Medical training was taken at the Long Island College of Medicine, from which institution he graduated in 1920.

The following year he started practicticing and was at times connected with the Flower, Israel Zion and Ocean Hill Hospitals. He was certificated by the American Board of Otolaryngology in 1937 and the following year was elected to Fellowship in the American Academy of Ophthalmology and Otolaryngology. He was also a fellow of the American Medical Association. Locally he was affiliated with the King County Medical Society.

Dr. Smith died October 28, 1943, and is survived by his wife, a son and a daughter.

HARMON LAMONTTE STANTON

Dr. Stanton, 50 year old Evansville, Ind., physician, was a graduate of the State University of Iowa College of Medicine, Iowa City, in 1920. Certified by the American Board of Otolaryngology, he was elected to Fellowship in the American Academy of Ophthalmology and Otolaryngology in 1928. He was a past president of the Vanderburgh County Medical Society, and belonged also to the American Medical Association and the Indiana State Medical Association.

In Evansville he served as president of the city board of health and was on the staffs of Deaconess and St. Mary's Hospitals. Death occurred on April 23, 1944.

CHARLES MIDWOOD STILES

Dr. Stiles was born in Shepton-Mallet, England, in January, 1866. His father, a Church of England clergyman, was headmaster of one of the largest boys' public schools in England. Dr. Stiles obtained his education in this school and at Oxford in Cambridge. In 1898 he received his degree of doctor of medicine from the Medico-Chirurgical College of Philadelphia and upon graduation he began the practice of ophthalmology in that city.

During World War I, Dr. Stiles served as a captain in the medical corps of the U. S. Army. He was a founder and member of the staff of the Frankford Hospital, Philadelphia. He was a member of the Medical Society of the State of Pennsylvania and in 1908 he became a Fellow of the American Academy of Ophthalmology and Otolaryngology.

His death occurred January 27, 1944.

GEORGE HAMILTON WALKER

Dr. Walker was born in Missouri, January 5, 1880. He grew up in Pawnee City, Neb., and was graduated from the State University in 1902. He spent a year as Nebraska state chemist and one year in the government assay office at Breckenridge, Colo. He then entered medical school at the University of Minnesota, interned at St. Luke's Hospital in St. Paul, and in 1909 he began general practice in Fairfax, Minn., where he remained until 1922. After leaving Fairfax, Dr. Walker spent a year in London and Vienna making a special study of the eye, ear, nose and throat. In 1926 he moved to Winona, Minn., and became associated with the Winona Clinic.

Dr. Walker was a fellow in the American College of Surgeons and a member of the Minnesota Academy of Ophthalmology and Otolaryngology. He became a Fellow in the American Academy of Ophthalmology and Otolaryngology in 1927. He was a member of the board of trustees of the local Methodist church.

Dr. Walker died July 2, 1943. His wife, Louise Feton Walker, and a son, Lt. George L. Walker of the United States Medical Corps, survive him.

SCIENTIFIC EXHIBITS

O. E. Van Alyea, M.D., Chairman
Chicago, Illinois
Georgiana Dvorak-Theobald, M.D.
Consultant
Chicago, Ill.

Exhibitor:
J. E. ASH, Col., M.C., U.S.A.
Army Medical Museum
Washington, D. C.
BY INVITATION
Co-Exhibitors:
HELENOR CAMPBELL WILDER
BY INVITATION
BENJAMIN RONES, M.D.
and
H. M. GOLDMAN, Lt., D.C., A.U.S.
BY INVITATION

I. Traumatic Injuries of the Eye in Soldiers

II. Tumors of the Ear, Nose and Throat

I. The pathologic changes resulting from various types of traumatic injuries to the eye and from retained foreign bodies are illustrated by photographs, photomicrographs and statistical charts. Particular attention has been paid to the developmental stages of the lesions.

II. The exhibit will consist of color transparencies and other photographs illustrating the microscopic appearances of interesting tumors which have been submitted to the Registry of Otolaryngic Pathology at the Army Medical Museum.

BIOLOGICAL PHOTOGRAPHIC ASSOCIATION
Chicago, Ill., Chapter
J. Garner, M.D., President
BY INVITATION

Medical Photography

The exhibit includes examples of medical photography of special interest to the ophthalmologist and otolaryngologist. Some of the equipment used for photographic and reproduction purposes will be demonstrated.

Exhibitor:
HOWARD A. CARTER, B.S. in M.E.
BY INVITATION
Chicago, Ill.
Council on Physical Medicine
American Medical Association

Lip Reading

The hard of hearing may be rehabilitated by selection and use of a suitable hearing aid, or by lip reading, or both. Present day practice is to supplement the hearing aid by lip reading, and vice versa. In cases of severe loss of hearing and in case of total deafness lip reading is probably the most satisfactory and convenient method for the patient to make contact with the outside world. Advice on the subject and names of places for obtaining instruction may be obtained.

Co-Exhibitors:
KENNETH DAY, M.D., Pittsburgh, Pa.
SYLVAN GOLDBERG, M.D.
Los Angeles, Calif.
HOWARD HOUSE, M.D.
Los Angeles, Calif.
MARVIN JONES, M.D., New York, N. Y.
CHARLES KINNEY, M.D.
Cleveland, Ohio
JULIUS LEMPERT, M.D.
New York, N. Y.
JAMES H. MAXWELL, M.D.
Ann Arbor, Mich.
JACOB MAYBAUM, M.D.
New York, N. Y.
PHILIP MELTZER, M.D., Boston, Mass.
ROBERT MOORHEAD, M.D.
Brooklyn, N. Y.
GEORGE E. SHAMBAUGH, JR., M.D.
Chicago, Ill.
J. MORRISETT SMITH, M.D.
New York, N. Y.
LaVERNE SPAKE, M.D.
Kansas City, Kan.
HENRY WILLIAMS, M.D.
Rochester, Minn.
DOROTHY WOLFF, Ph.D.
Boston, Mass.
BY INVITATION

Functional Labyrinthine Surgery

Results of histological investigation of both humans and animal tissues from a

research study of the Fenestration Operation. Cadaver specimens and drawings showing the surgical technics of the Lempert Fenestra Nov-Ovalis operation for clinical otosclerosis, the Kenneth Day operation for Meniere's Disease, the Lempert Decompression Operation for Meniere's Disease and the anatomic details of the perilymphatic and endolymphatic labyrinth. Charts showing indications and end results obtained with functional labyrinthine surgery. Evaluation of the various means employed to secure a maximum of permanently open Fenestra Nov-Ovalis and a minimum of permanently damaged endolymphatic labyrinths.

Exhibitor:

SAMUEL FOMON, M.D.

BY INVITATION

New York, N. Y.

Auspices of the American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery

Otolaryngologic Plastic Surgery

1. Plaster and wax models illustrating various phases of operative technic in plastic procedures.
2. 33 diagrammatic illustrations demonstrating technic of typical rhinoplasty.
3. Selectroslide projection of plastic procedures of interest to the otolaryngologist.
4. X-ray plates showing fractures of the nose and malarzygomatic compound.
5. Methods employed in making a diagnosis.
6. Models showing plastic reconstruction of septal deformities.
7. Animated motion pictures.

Exhibitor:

JAMES S. GREENE, M.D.

National Hospital for Speech Disorders
New York, N. Y.

Illuminated photographs, taken at the National Hospital for Speech Disorders, illustrate diagnostic and therapeutic procedures carried out in a model speech clinic. Various oral and laryngeal anomalies are shown, and records demonstrate their effects on the speech function.

Exhibitor:

J. Q. GRIFFITH, JR., M.D.

BY INVITATION

University of Pennsylvania
Philadelphia, Pa.

Co-Exhibitor:

WILFRED E. FRY, M.D.

Papilledema; Mechanism and Clinical Applications

Exhibit illustrating a series of experiments in which papilledema was produced in laboratory animals under carefully controlled conditions. These experiments support the following hypothesis: Papilledema results when pressure is exerted on the central vein of the retina where it traverses the perineural optic space. Under normal conditions the perineural optic space is continuous with the subarachnoid space, and any condition causing increased pressure in the cerebrospinal fluid will cause papilledema. However, if for any reason the perineural optic space is blocked and is not continuous with the subarachnoid space, increased pressure in the cerebrospinal fluid will not cause papilledema. Various pathologic states are reviewed, including brain tumor, brain abscess, subarachnoid hemorrhage, meningitis of bacterial origin, encephalitis, hydrocephalus, sinus thrombosis, intracranial aneurysm, cerebral trauma, congenital malformations and hypertension.

Exhibitor:

GEORGE P. GUIBOR, M.D.

Children's Memorial Hospital
Chicago, Ill.

Synkinetic Overaction of the Inferior Oblique Muscle

Overaction of the inferior oblique muscle associated with a paresis of the contralateral superior rectus muscle or a paresis of the homolateral superior oblique muscle is well recognized. On the contrary overaction of the inferior oblique resulting from supranuclear lesions and from paresis of a lateral rectus muscle or from a paresis of an internal rectus muscle is not often recognized. This overaction is a phenomena made possible by synkinetic connections of the inferior oblique muscle. The synkinetic overaction should be diagnosed because such a diagnosis suggests that the treatment of the hypertropia or hyperphoria is not the treatment of the vertical disturbance but of the cause of the vertical disturbance, which may be a horizontal motor defect.

Exhibitor:
A. C. HILDING, M.D.
Duluth, Minnesota

Factors for Safety in Surgery for Cataract

This exhibit demonstrates: (a) experiments showing comparative physical properties of cornea and sclera, especially as to resilience, (b) experiments on eyes of animals and man showing the mechanics of iris prolapse, (c) two types of cataract operations based upon these experiments, (d) a series of cases operated upon by each of the two methods with results and complications.

Exhibitor:
A. R. HOLLENDER, M.D.
University of Illinois College of Medicine
Chicago

Co-Exhibitors:
HANS BRUNNER, M.D.
PAUL B. SZANTO, M.D.
BY INVITATION

The Nasopharynx
Anatomic, Pathologic and Clinical Aspects

Exhibit showing the anatomy with diagrams, drawings and actual specimens; the pathology with gross specimens and photomicrographs, and the clinical aspects with drawings of diagnostic and therapeutic technic.

Co-Exhibitors:
ISOBEL JANOWICH
and
ELEANOR BROWN MERRILL
BY INVITATION
National Society for the Prevention of Blindness
New York, N.Y.

Progress in Prevention of Blindness

The exhibit will consist of graphic charts showing advances in various phases of the Society's program, with special emphasis on industrial ophthalmology and its relation to conservation and utilization of eyesight in industry.

JOINT COMMITTEE ON INDUSTRIAL OPHTHALMOLOGY
Albert C. Snell, M.D., Chairman

Eye Protection Devices

This is an exhibit of industrial goggles and other eye protective devices, their construction, uses, and their relation to specific industrial occupations and operations.

Exhibitor:
V. H. KAZANJIAN, M.D.
Massachusetts Eye and Ear Infirmary
Boston, Mass.

Plastic Clinic

Reconstruction After Radical Operation for Osteomyelitis of the Frontal Bone

Various methods of restoring the lost frontal bone are to be shown. The problems that are encountered, together with the various materials at the disposal of the operator will be discussed. There will be a series of models, pictures and roentgen rays to illustrate the technic.

Exhibitor:
PETER C. KRONFELD, M.D.
Illinois Eye and Ear Infirmary
University of Illinois
Chicago
Co-Exhibitor
HENRY N. RICCI, M.D.
BY INVITATION

Testing and Rehabilitation of Schiotz Tonometers

A Checking Station in Action

The exhibit is sponsored by the Committee on Standardization of Tonometers of the American Academy of Ophthalmology and Otolaryngology. The procedure of checking tonometers devised by Posner and Schoenberg will be shown in detail. Some of the mechanical defects or deviations from the standard can be rectified by the manufacturer. Others such as improper weight of the plunger or foot plate assembly can be corrected by providing new conversion tables for the particular Schiotz tonometer. In this way a great many used Schiotz Tonometers can be rehabilitated.

Exhibitor:
PETER C. KRONFELD, M.D.
Illinois Eye and Ear Infirmary
University of Illinois
Chicago
Co-Exhibitor
H. I. McGARRY, M.D.
and
J. S. HAAS, M.D.
BY INVITATION

Operations for Glaucoma

An Investigation into Their Mode of Functioning

By various clinical methods an attempt has been made to determine the way in which the elevated intraocular pressure in the glaucomas is controlled by the four

classical operations, the iridectomy, the corneosclerectomy, the iridencleisis and the cyclodialysis.

Exhibitor:
M. L. LEWIN, Capt., M.C., A.U.S.
Bushnell General Hospital
Brigham City, Utah

Gunshot Wounds of Face as Seen in a Maxillo-Facial Center of the U. S. Army

The exhibit will include photographs and drawings showing the activities of a maxillo-facial center in handling wounds and deformities of face due to explosives (bullets, bomb fragments, land mines, etc.). Surgical management of the following injuries will be depicted: soft tissue losses, defects in facial skeleton, fractures of the mandible with delayed union or nonunion, fractures of the maxillary compound, contractures of oral, nasal and orbital mucosa. It will also include wax models showing comminuted fractures of mandible and maxilla and illustrating bone grafts and various splinting devices.

Exhibitor:
MURIEL McLATCHIE
BY INVITATION
Massachusetts General Hospital
School of Medical Illustration
Boston, Mass.

Illustrative Anatomy

The School of Medical Illustration has partially devoted itself, in this exhibit, to drawings which would interest those individuals whose particular field is the eye, ear, nose and throat. It is to you we offer, for comment, our teaching drawings which include operative and scopic work, the sinuses, introduced in an "X-ray technic," the normal eye and ear in water color, and the skeletal head and neck. We have also demonstrated, for your further perusal, three life-size skeleton illustrations with a detailed life-size half-tone manikin and surgical, medical and pathological drawings.

Exhibitor:
SAMUEL J. MEYER, M.D.
Illinois Eye and Ear Infirmary
University of Illinois
Chicago
Co-Exhibitor:
PAUL STERNBERG, M.D.
BY INVITATION

The Surgical Management of Primary Glaucoma with the Aid of Gonioscopy and Biomicroscopy

Gonioscopy and biomicroscopy can be of limited aid in the surgical management of the various types of primary glaucoma. These clinical instruments of investigation present evidence with which the ophthalmologist may better evaluate the type, site, and means to the surgical approach of the individual glaucoma problem.

Exhibitor:
DARIUS GRAY ORNSTON, M.D.
BY INVITATION
Jefferson Medical College
Philadelphia, Pa.

Color Photographs of Value in Teaching

A group of color photographs displaying (1) various pathological states of the ears, nose and throat and (2) a method of determining the health of the nasal mucosa by observing the rapidity with which it cleanses itself of micraform crystals.

Exhibitor:
LE ROY A. SCHALL, M.D.
Massachusetts Eye and Ear Infirmary
Boston, Mass.

Cancer of the Larynx

An educational exhibit showing types of laryngeal cancer and anatomical specimens.

Exhibitor:
MARK J. SCHOENBERG, M.D.
National Society for the Prevention of Blindness
New York, N.Y.

Glaucoma

Graphic presentation of the following aspects of glaucoma: pathology; gonioscopy; scotometry; perimetry; pupillography; geographic distribution of glaucoma; special glaucoma clinics and services; and the general practitioner's part. Prepared by a number of the members of the Committee on Glaucoma of the National Society for the Prevention of Blindness, and other cooperating ophthalmologists.

Exhibitor:
J. D. SINGLETON, M.D.
Southwestern Medical College
Dallas, Texas

Anatomical Variations of the Temporal Bone: Surgical Anatomy

The exhibit consists of approximately 44 dissections of the temporal bone showing a wide range of anatomical variations. Unusual tracts and cell groups are shown and described. Several specimens show dense septa separating various cell groups. Other specimens show well done and poorly done operations that are commonly performed upon the mastoid.

Exhibitor:
MAURICE F. SNITMAN, M.D.
University of Illinois College of Medicine
Chicago
Co-Exhibitors:
EDMUND V. PELLETTIERE, M.D.
PIERCE W. THEOBALD, M.D.

BY INVITATION

Carcinoma of the Larynx

Improved prognosis of carcinoma of the larynx depends very largely on (a) a more comprehensive study of the extension of the disease, and (b) a more detailed investigation of its histopathology.

This exhibit will consist of:

1. Serial histologic sections of normal larynges of persons of different age groups.
2. Serial histopathologic sections of larynges removed for carcinoma of the larynx.
3. Serial histopathologic sections of vocal cords with malignant involvement, removed by thyrotomy.
4. A proposed classification of carcinoma of the larynx, illustrated with microphotographs.

Exhibitor:
O. E. VAN ALYEA, M.D.
University of Illinois College of Medicine
Chicago

Treatment of Acute Infections of the Nose and Sinuses

Treatment of acute nasal and sinus infections is undergoing constant change at the present time and improved methods are being adopted from time to time to conform to our ever increasing knowledge of nasal physiology and the histopathology of respiratory mucosa. In this exhibit modern medical and surgical procedures are described and the basis for these procedures is presented.

Exhibitor:
ALAN C. WOODS, M.D.
Johns Hopkins Hospital
Department of Ophthalmology
Baltimore, Md.

This is an art exhibit of the more recent work done by Mrs. Burgess and is a collection of paintings and drawings illus rating various pathological conditions of the eye.

TECHNICAL EXHIBITS

Grateful appreciation is expressed to the Technical Exhibitors

Acousticon Division, Dictograph Sales Corp, New York, N. Y.
Booths 36 and 37

The Acousticon exhibit will be devoted to the new Cooperative Medical Service Plan, whereby physicians can participate directly in recommending and fitting hearing aids. There will be exhibited the new Acousticon Speech-Hearing Test which embodies all the basic speech sounds of all pitch and loudness ranges in our language. This test equips the physician with an **objective technic**, by means of scored results, to determine

1. the pattern and degree of speech-hearing deficiency
2. the amount and pattern of corrective amplification required
3. the amount of speech-hearing restoration furnished by a hearing aid.

The exhibit will also feature the new Acousticon Speech-Hearing Re-education Program for re-educating the hard of hearing to enable them to regain their lost ability to hear and use speech sounds.

American Optical Company, Southbridge, Mass. Booths 43 to 50

American Optical Company of Southbridge, Mass., cordially invites everybody to visit its booth where AO ophthalmological equipment will be shown. Also on exhibit will be scientific instruments made by the Spencer Lens Company, AO's scientific instrument division. AO representatives will be glad to discuss and demonstrate this equipment, some of which, due to war demands, is only available in limited quantities. Another

feature of AO's exhibit will be a series of four oil paintings illustrating historical phases in the correction of eyesight. Painted by Herbert Morton Stoops, noted American artist, the four pictures depict the transition from the crude "fit yourself" method of "buying specs" fifty years ago to the professional ophthalmic services of today.

Barnett Optical Laboratories, Chicago, Ill. Booth 19

This year the Barnett Optical Laboratories will exhibit the miniature rechargeable flashlight storage battery which replaces two size D dry cells. It is recharged by means of a small 115 volt AC charger. The battery which has been used in industrial fields for many years is estimated to outlast 400 dry cells.

A series of 35 mm. kodachrome transparencies of various eye lesions, with brief histories for reference and instruction purposes, will also be on display.

Bausch & Lomb Optical Co., Rochester, N. Y. Booth 70

Ophthalmologists who attend the annual convention of the Academy will have an opportunity to get first-hand information about the Bausch & Lomb Industrial Vision Service, a plan which enables industrial management to study and utilize more completely the visual skills and capacities of employees. By means of the Ortho-Rater, a new Bausch & Lomb instrument, and a visual fact-finding system, the need for professional visual care by many employees on many jobs is revealed, and to a greater degree than has been suspected. Much of the early research in connection with the Industrial Vision Service was done by Hedwig S. Kuhn, M.D. The results of Dr. Kuhn's work are described in her new book, "Industrial Ophthalmology."

At the meeting last year, Academy members saw for the first time the new Bausch & Lomb publication, "The Human Eye in Anatomical Transparencies." From this book, which incidentally is now in its second printing, original kodachrome slides of the Gladys McHugh anatomical paintings have been made. These will be on display at the 1944 convention.

The Bausch & Lomb exhibit will also include modern ophthalmic instruments for refraction and diagnosis. The delivery of some Bausch & Lomb instruments is limited to the military services, but because of their particular interest to Academy members vitally concerned with the health of a nation at war, every effort will be made to have a variety on hand for inspection.

Bausch & Lomb Affiliated Distributors
Colonial Optical Company, New York
McIntire, Magee and Brown Company, Philadelphia
Riggs Optical Company, Chicago and San Francisco
Southeastern Optical Company, Richmond
White-Haines Optical Company, Columbus

Booths 11, 12 and 13

This exhibit will feature THE INDUSTRIAL PRESCRIPTION GOGGLE in all of its phases. An elaborate and instructive display of all types of industrial ophthalmic prescription lenses including **hardened myodisc** lenses for high myopia, **hardened bifocal** and **single vision** lenses in white and absorptive types will be shown. Special types of goggle frames for specific uses such as welding, acid protection, etc., will supplement this interesting and unusual display.

A complete Bausch & Lomb ophthalmic instrument array together with the new major amblyoscope known as the Wottring Troposcope will complete the exhibit.

Belgard, Inc., Chicago, Ill. Booth 38

Belgard, Inc., will exhibit their line of refracting accessories, with Austin Belgard in charge.

Included in the exhibit will be the firm's "Opticheck," a new wartime product for centering lenses, locating axes and measuring prismatic power. This device is made without the use of critical materials and combines a remarkable degree of efficiency with low cost. Also to be shown are a new illuminated portable Visual Acuity Chart, especially suitable for visiting school nurses, bedside refracting and auxiliary refracting rooms, and a Near Point Visual Acuity Chart with illiterate "E" symbol for quantitative measurement of vision at the near point.

Mr. Belgard extends a cordial invitation to Academy members and visitors to drop in.

Belgard-Spero, Inc. (The House of Vision), Chicago, Ill. Booth 69

The House of Vision, with headquarters in Chicago, Ill., and plants located in Aurora, Ill.; Muskegon, Mich.; Minneapolis, Minn.; Milwaukee, Wis.; and Oak Ridge, Tenn., will have booth number 69, where of course all who attend the meeting will be most heartily welcomed.

This company, noted for its contribution of a number of valuable optical aids, will not have a merchandise display, but hopes to have ready some things of real interest to the ophthalmologist.

Bell & Howell Company, Chicago, Ill. Booth 17

We will exhibit motion picture cameras, projectors and accessories; also 2′ x 2′ slide projectors and sound motion picture projectors.

Literature will be available on our extensive 16 mm. film library.

Camel Cigarettes, New York, N. Y. Booths 30 and 31

CAMEL Cigarettes will exhibit large detailed photographs of equipment used in compartive tests of the five largest-selling brands of cigarettes. Dramatic visualization of nicotine absorption in the human respiratory tract from cigarette smoke will be demonstrated. International news with the CAMEL Cigarette Trans-Lux "Flash Bulletins" may be seen while enjoying a supply of slow-burning CAMEL cigarettes.

Cameron Surgical Specialty Company, Chicago, Ill. Booth 25

Introducing the new Cameron bronchoscopic, esophagoscopic and laryngoscopic instruments. Also see the Binocular Prism Loupe, the Mirrolite, 5-in-1 Red Free Retino-Ophthalmoscope and other latest developments in electrically lighted diagnostic and operating instruments for all parts of the body. You will also be interested in our Radio Frequency Knives, Coagulators and Electrified Snares.

Contact Lens Service, Inc., New York, N. Y. Booth 66

Contact Lens Service will offer a most interesting exhibit, which will include eye moldings at various intervals during the sessions; also, a demonstration of fitting and adjusting lenses. All the materials needed for contact lens prescribing and fitting will be on display.

F. A. Davis Company, Philadelphia, Pa. Booth 18

At booth 18 you may examine the newest books on Ophthalmology and Otolaryngology. Also, if, like many other physicians, you are today being forced because of conditions into doing practice outside of your specialty, you will be interested in examining the following publications: Loewenberg — Medical Diagnosis and Symptomatology; Smith—Sulfonamide in Medical Practice; Judovich & Bates—Segmental Pain; Robertson—Diagnostic Signs, Reflexes and Syndromes; Blum — Dietetics; Mullen — Handbook of Treatment; Willard—Digestive Disorders; Bortz—Diabetes; Reimann—Treatment in General Medicine; Dutton — Headache and Head Pains; Murphy—Acute Medical Conditions.

DeVilbiss Company, Toledo, Ohio Booth 65

The evolution of nose and throat solutions to sulfonamide compounds, plus research conducted by pharmaceutical manufacturers that has proven spray application more effective, makes plastic atomizers the most advanced instruments for scientific application of nose and throat solutions.

A complete line will be exhibited at booth No. 65. Mr. L. H. Smock, in charge of the DeVilbiss exhibit, will welcome the opportunity to demonstrate and explain the many outstanding features of this line.

Doho Chemical Corporation, New York, Montreal, London Booth 23

ANIMATED PATHOLOGICAL EAR EXHIBIT — The Auralgan Exhibit consists of a model of the human auricle four feet high, together with a series of 24 three-dimensional ear drums modeled under the supervision of outstanding otologists. Each of these drums depicts a different pathologic condition based upon actual case observation, and prepared, insofar as possible, with strict scientific accuracy so as to be highly instructive and interesting to all physicians.

Graybar Electric Company, Inc., New York, N. Y. Booth 5

At the Academy Convention in Chicago, October 8-12, will be displayed the latest scientific equipment designed by the Bell Telephone Laboratories, manufactured by the Western Electric Company and distributed through the 86 offices of the Graybar Electric Company to dealers throughout the United States.

Included in this exhibit will be all models of Western Electric Hearing Aids.

Grune & Stratton, Inc., New York, N. Y. Booth 21

The GRUNE & STRATTON exhibit will comprise an established list of titles by foremost writers presenting the latest advances in research and practice. Among them, the visitors will find the well-known book by Drs. Fischer and Wolfson on THE INNER EAR as well as the most recent publication by Drs. Spiegel and Sommer on the NEUROLOGY OF THE EYE, EAR, NOSE, AND THROAT. Many other books of general interest will also be exhibited.

Guild of Prescription Opticians of America, Inc., Philadelphia, Pa. Booth 6

The Guild exhibit will be institutional in character showing the general work of Guild opticians: the education of the public to the importance of eye care as it is rendered by the ophthalmologist and the necessity for periodical examination; the overcoming of the indifference or the disregard of danger signals of eye care; and, in the interests of public eye care, to encourage complete collaboration between the eye physician, who examines, refracts, and prescribes, and the dispensing optician (ophthalmic dispenser) who interprets, compounds, fits, and services prescriptions for the correction of vision.

The aim of the Guild of Prescription Opticians is to advance the science of ophthalmic optics through the development of a country-wide ethical dispensing optical service, which comprehensively meets the needs of the eye physicians and their patients; and to educate the public to the fact that the Eye Physician-Guild Optician type of eye service truly renders the most efficient form of eye care.

Invisible Lens Service, Buffalo and Syracuse, N. Y.; Cleveland, Ohio; Stroudsburg, Pa. Booth 39

Our services on the molding, fitting, and technical work on Invisible Lenses give the ophthalmologist a complete service for his patients.

We will display complete equipment, and our technician in charge at this convention will explain in detail our services or will call at your office should you be located in Ohio, New York or Pennsylvania.

Vision has been restored on conical corneas, corneal scars, high myopes, and cataract cases. Consult us on your difficult refraction cases.

Keystone View Company, Meadville, Pa. Booth 32

During the past year revised manuals for the BU and AN Series exhibited at the American Academy of Ophthalmology and Otolaryngology last year have been published. Likewise, in response to many users of the Zeta Home-Training Unit, a Home-Training Unit with greater base-out and base-in requirements has been completed. This is called the Eta Unit. These and other new developments will be available for examination at the Keystone exhibit.

Lea and Febiger, Philadelphia, Pa. Booth 77

Lea and Febiger will exhibit at the American Academy of Ophthalmology and Otolaryngology a collection of new works and new editions. Among the new editions will be: Spaeth's "Principles and Practice of Ophthalmic Surgery"; Gifford's "Handbook of Ocular Therapeutics"; Gray's "Anatomy of the Human Body"; Ballenger's "Diseases of the Nose, Throat and Ear, Medical and Surgical"; Ballenger's "Manual of Otology, Rhinology, and Laryngology"; Bell's "Textbook of Pathology"; Boyd's "Textbook of Pathology"; Craig and Faust's "Clinical Parasitology"; Gershenfeld's "Urine and Urinalysis"; Kovacs' "Manual of Physical Therapy"; Kraines' "Therapy of the Neuroses and Psychoses"; Levinson and MacFate's "Clinical Laboratory Diagnosis"; Ormsby and Montgomery's "Diseases of the Skin"; Rhinehart's "Roentgenographic Technique"; Rowe's "Elimination Diets and the Patient's Allergies"; Simmons and Gent-

skow's "Laboratory Methods of the U. S. Army"; Speed's "Textbook of Fractures and Dislocations." New works which will be shown are: Lewin's "Backache and Sciatic Neuritis"; Babcock's "Principles and Practice of Surgery"; Donaldson's "Surgical Disorders of the Chest."

Mager & Gougelman, Inc., Chicago and New York Booth 35

Plastics bring a new technic to the making of artificial eyes. These plastic prostheses are especially valuable in cases of socket or globe deformation. They do not shatter, crack, become rough or discolored.

The new Woods' Plastic Splints employed in lime, lye and acid burns to prevent adhesions are superior to the method of using lead plates or cellophane.

Maico Company, Inc., Minneapolis, Minn. Booth 42

MAICO EXHIBIT OF NEW HEARING DEVELOPMENTS — Visitors at the Maico Booth may acquire a quick understanding of Army-Navy precision hearing tests while discovering any hidden defects in their own ears by checking their hearing against the unique Maico Tone-Color organ. This colorful unit, resembling a pipe organ in rainbow colors, enables the visitor to measure his own hearing for the audible spectrum in terms of the visible spectrum.

The latest tiny vacuum tube hearing aids, individually fitted to compensate for hearing defects as revealed by audiometric test, are also shown.

Medical Film Guild, New York, N. Y. Booth 71

Medical Film Guild emphasizes its talking papers in this year's program of "MEDICAL FILMS THAT TEACH." Hospital and Medical Society program chairmen, now faced with depleted staffs because of the war emergency, who desire educational material for their meetings, find that Medical Film Guild's motion picture film textbooks answer that important problem. Through grants for postgraduate instruction, these films are available at no charge to any hospital, medical society meeting or the medical services connected with the Armed Forces of the United States. Exhibition is also included at no charge under this plan. Subjects available are:

Inguinal Hernioplasty
Asphyxia Neonatorum
Nonoperative Treatment of Paranasal Sinusitis
Otitis Media in Pediatrics
A Clinic on Acute Mastoiditis
Otoscopy in the Inflammations
A Clinic on Sigmoid Sinus Thrombosis
A Clinic on Petrositis with Meningitis
Amebiasis and Its Treatment
A Clinic on Otitic Purulencies
Pharmacology of Respiratory Stimulants

William S. Merrell Company, Cincinnati, Ohio Booth 72

The Merrell exhibit will feature the important new vasoconstrictor, Solution VONEDRINE WITH CEEPRYN, which provides prolonged nasal vasoconstriction without irritation, secondary congestion or central nervous stimulation. Combining the new vasopressor amine, Vonedrine, with the nonmercurial germicide, Ceepryn, in an aqueous isotonic solution with pH adjusted to 5.5, this self-sterilizing solution for application as spray, drop or tamponade is safe, effective and pleasant to use. Complete literature and samples are available at the Merrell booth.

C. V. Mosby Company, St. Louis, Mo. Booth 22

Among the publications to be displayed by the C. V. Mosby Company at this year's meeting of the American Academy of Ophthalmology and Otolaryngology will be the new Kuhn "Industrial Ophthalmology" and Bellows "Cataract and Anomalies of the Lens." A cordial invitation is extended to all attending the convention to visit Booth No. 22 and inspect these and the many other medical texts which will be on display.

V. Mueller and Company, Chicago, Ill. Booths 51 to 57

The exhibit of V. Mueller & Company will include a complete line of fine, hand made, stainless steel instruments for eye surgery, as well as instruments for ear, nasal and plastic work. Several interesting new developments will be presented, along with the latest improvements in Mueller surgical equipmnt for the eye-ear-nose-throat practitioner.

New Jersey Optical Company, Irvington, N. J. Booth 4

Emphasizing their slogan, "One Line Only — The Finest," the New Jersey Optical booth at the October Convention will be of special interest. This concern has always, since its inception, guaranteed all frames and mountings to contain not less than 50/1000 Fine Gold — well over the minimum specified by law. They feel that this definite specification gives tangible evidence of less obvious, but equally important, standards of workmanship and quality.

While the New Jersey Optical Company manufactures a complete line of frames and mountings, their leaders are E-Z-Mont — a 4-point suspension, hidden top-wire mounting and Wils-edge — an entirely unique mounting which suspends lenses from a grooved top-wire that holds them firmly in adjustment, without screws or holes.

We feel sure that a visit with the New Jersey Optical people will prove worth while.

Novocol Chemical Mfg. Company, Inc., Brooklyn, London, Toronto, Buenos Aires, Rio de Janeiro Booth 60

For 33 years the company has devoted its energies to the development of safer and more potent local anesthetics. We suggest you investigate the reasons for the popularity MONOCAINE solutions enjoy with the medical profession. At Booth 60 the improved method for the administration of local anesthetics is on display.

Obrig Laboratories, Inc., New York, N. Y. Booth 16

Contact lenses are being used more and more to rehabilitate persons who have had corneal injuries. After the war there will be a greater need for these devices to help restore vision to men who have received corneal wounds.

The Obrig Laboratories will exhibt contact lenses, gonioscopic lenses, microscopic lenses, tinted lenses and the materials for making impressions of the eyes, as well as the equipment for adjusting the fitting of contact lenses.

Many leading ophthalmologists consider contact lenses to be "the glasses of the future."

The all-plastic unbreakable contact lens is 60 per cent lighter than the glass lens; it is inert and easily adjustable and can be ground and polished to any correction.

Optical Specialties Corporation, New York, N. Y. Booths 75 and 76

The Optical Specialties Corporation is established to introduce new products and services which will contribute to scientific and professional progress. It is the policy of the company to undertake the distribution of a new product only after consultation with men of recognized standing in the profession, and an important function of the corporation is to stimulate the development of devices and procedures for which a need exists. The Optical Specialties Corporation welcomes suggestions from the profession and from inventors as to suitable products and services which the company may develop.

Philip Morris and Company, Ltd., New York, N. Y. Booth 64

Philip Morris & Company will demonstrate the method by which it was found that Philip Morris Cigarettes, in which diethylene glycol is used as the hygroscopic agent, are less irritating than other cigarettes. Their representative will be happy to discuss researches on this subject, and problems on the physiological effects of smoking.

George P. Pilling and Son Company, Philadelphia, Pa. Booths 1, 2 and 3

We are devoting almost all our manufacturing facilities to produce urgently needed surgical instruments for the Army and Navy, and therefore we will not be able this year to show a complete line of our specialties as in the past. There will be prominent gaps in the line.

However, extra special efforts are being made to assemble an interesting exhibit, and we feel confident it will be worth your inspection as it always has been in the past.

We hope our friends, therefore, will visit us and as usual every possible effort will be made to serve them.

Precision Contacts, Minneapolis, Minn. Booth 67

There will be a display of Precision all-plastic contact lenses and equipment used to fit them. A new improved Precision molding shell will be shown. Complete information on availability of contact lens fitting instruction will be sup-

plied. A new and up-to-date instruction manual, "The Technic of Fitting Contact Lenses" by A. L. Anderson may be inspected at the booth.

Ritter Company, Inc., Rochester, N. Y. Booths 61, 62 and 63

Ritter Co., Inc., invites you to see demonstrated the Ritter Ear, Nose and Throat Unit, Ritter Motor-Driven Treatment Chairs, Ritter Shockproof X-ray Unit, Ritter Fluorescent Examination and Operating Lights, Ritter Surgical Cuspidor (Aspirator Type), Ritter Examination and Operating Stools, Ritter Hydromatic Sterilizers, Ritter Automatic Air Compressors and Ritter Bone Surgery Engine.

Schenley Research Institute, New York, N. Y. Booths 73 and 74

An interesting and informative exhibit portraying the clinical indications and administration of Penicillin **Schenley**; also showing its manufacture, standardization, and testing. Special attention is given to the use of Penicillin **Schenley** in ophthalmology and otolaryngology.

Shuron Optical Company, Inc., Geneva, N. Y. Booths 40 and 41

More and more ophthalmologists are realizing that Shuron Shurset Rimway Ful-Vue mountings are ideal for holding ophthalmic lenses in permanent alignment with maximum security and a minimum of strain. The manufacture, distribution and use of nearly four million Shursets in little more than four years prove that the suspended lens principle is right.

A cordial invitation is extended by Shuron to those Academy members who are not familiar with the Shurset mounting and its adaptations to visit our booths for a quick, practical demonstration.

Smith, Kline and French Laboratories, Philadelphia, Pa. Booth 8

Come to booth 8 for information on how the physician may save himself time and trouble and spare the patient prolonged visual disability by using the "Paredrine" technic in refractions. Also, we will gladly explain the local use — both medical and surgical — of "Micraform" sulfathiazole in rhinologic and otologic practice. "Micraform" preparations include "Paredrine" — Sulfathiazole Suspension, "Micraform" Sulfathiazole Suspension, 5 per cent, and "Micraform" Sulfathiazole Suspension, 20 per cent.

Soft-Lite Lens Company, Inc., New York, N. Y. Booths 9 and 10

Ophthalmologists in increasing numbers find that the neutral absorption afforded by Soft-Lite Lenses gives patients greater comfort when their refraction indicates the need of protection from natural or artificial over-brightness. Soft-Lite Lenses are available in five densities in the finest single vision and bifocal forms — all made by Bausch and Lomb exclusively for the Soft-Lite Lens Company, Inc., New York, Toronto, London.

C. M. Sorensen Company, Inc., New York, N. Y. Booth 14

The best available equipment in our line for ear, nose and throat work will be on display for your inspection. We look forward to greeting old friends and making new ones at this always interesting meeting."

Frederick Stearns and Company, Detroit, Mich. Booths 26 and 27

A cordial invitation is extended to all attending physicians to visit the Stearns booth where on display will be such items as Neo-Synephrine Topical and Surgical, Neo-Synephrine Ophthalmic — in which is used a new low surface tension vehicle, Appella Apple Powder, Gastric Mucin, and Vitamin Products.

Our professional representatives will be pleased to supply you with all the information on the use of these products which represent outstanding contributions to medical science developed in the Scientific Laboratories of Frederick Stearns and Company.

Surgical Mechanical Research
1903 Beverly Boulevard
Los Angeles 4, California
Booths 33 and 34

Uhlemann Optical Company, Chicago, Ill. Booths 58 and 59

Exhibit — PHYSICIANS QUALITY GLASSES. First to serve the ophthalmologist exclusively. Creators of new designs for modern eyewear.

Univis Lens Company, Dayton, Ohio
Booth 24

Increased interest in the low-reflection coating on spectacle lenses, as exhibited by Univis at the 1942 Academy, is responsible for a further display of coated lenses this year. The "ghost image" of a light viewed through the very low minus Rx's (−0.25 to −0.75) occasionally annoys the wearer to such an extent as to be intolerable. Low-reflection coating minimizes such a "ghost image" so that it is ignored by the wearer.

Also on display is expected to be the telescopic spectacle lenses which Univis hopes to have in production by October the 8th. At the beginning of the war, production was suspended on the 1.5x and 2.0x Univis 'scopes, but resumed production — either of the '41 models or of a still further improved design — is expected to be announced at the time of the Academy Convention.

Westwood Pharmacal Corporation, Buffalo, N. Y. **Booth 15**

WESTWOOD will exhibit WESTHIAZOLE E.N.T. and WESTHIAZOLE OPHTHALMIC — two sulfathiazole solutions representing an entirely new development in the field of local chemotherapy. WESTHIAZOLE E.N.T. and WESTHIAZOLE OPHTHALMIC contain 5 per cent sulfathiazole in solution, are water and exudate soluble, possess remarkable wetting properties and are relatively non-irritating. WESTHIAZOLE E.N.T. is sterile, has a pH of 6.2. WESTHIAZOLE OPHTHALMIC has a pH of 7.5. They are indicated for the treatment of infections of pneumococcic, staphylococcic and streptococcic origin. Drop in at our booth for generous trial supplies.

White Laboratories, Inc., Newark, N. J.
Booth 68

At the WHITE LABORATORIES' booth 68 you will find interesting copies of a series of publications under the general title "Diagnostic Aids to Vitamin Deficiency Conditions." Medical service representatives in attendance will be very glad to discuss these with you.

The latest clinical reports on results of the use of White's Vitamin A and D Ointment in the treatment of burns and various types of ulcers will also be available. This is a product which you will undoubtedly find of great interest.

Williams and Wilkins Company, Baltimore, Md. **Both 20**

The Williams and Wilkins Co. will exhibit its books and periodicals of interest in ophthalmology and otolaryngology, including new books and new editions of established books.

Max Wocher & Son Co., 609 College St., Cincinnati 2, Ohio **Booths 28 and 29**

Zenith Radio Corporation, Chicago, Ill.
Booth 7

"New smartness comes to the hearing aid by way of the new Zenith neutral color earphone and cord.

What modern styling did for eyeglasses, Zenith now does for the hearing aid! The visible parts blend beautifully with any complexion — are scarcely noticeable.

Come in — see the PROOF of new beauty, new convenience and durability, too, in this exclusive Zenith "First." The new Neutral-Color Cord will not fray, is perspiration proof, water-proof, kink-proof, and washable. Just wipe clean with a damp cloth. The result of more than two years of laboratory research — Zenith's Neutral-Color Earphone and Cord are now standard equipment — at no extra cost.

See this instrument demonstrated at the annual meeting to be held October 8-12 at the Palmer House in Chicago.

PAST OFFICERS OF THE ACADEMY

Date	President	First Vice-President	Treasurer	Secretary	Meeting Place
1896	Adolph Alt, M.D. St. Louis, Mo.	W. C. Pipino, M.D. Des Moines, Iowa	W. L. Dayton, M.D. Lincoln, Nebr.	Hal Foster, M.D. Kansas City, Mo.	Kansas City
1897	Adolph Alt, M.D. St. Louis, Mo.	W. C. Pipino, M.D. Des Moines, Iowa	W. L. Dayton, M.D. Lincoln, Nebr.	Hal Foster, M.D. Kansas City, Mo.	St. Louis
1898	B. E. Fryer, M.D. Kansas City, Mo.	J. E. Colburn, M.D. Chicago, Ill.	W. L. Dayton, M.D. Lincoln, Nebr.	Hal Foster, M.D. Kansas City, Mo.	Chicago
1899	J. E. Colburn, M.D. Chicago, Ill.	W. Scheppegrell, M.D. St. Louis, Mo.	W. L. Dayton, M.D. Lincoln, Nebr.	Thomas A. Woodruff, M.D. Chicago, Ill.	New Orleans
1900	W. Scheppegrell St. Louis, Mo.	M. A. Goldstein, M.D. St. Louis, Mo.	W. L. Dayton, M.D. Lincoln, Nebr.	Fayette C. Ewing, M.D. St. Louis, Mo.	St. Louis
1901	M. A. Goldstein, M.D. St. Louis, Mo.	H. W. Wurdemann, M.D. Milwaukee, Wis.	W. L. Dayton, M.D. Lincoln, Nebr.	W. L. Ballenger, M.D. Chicago, Ill.	Cincinnati
1902	C. R. Holmes, M.D. Cincinnati, Ohio	W. L. Dayton, M.D. Lincoln, Nebr.	Otto Stein, M.D. Chicago, Ill.	W. L. Ballenger, M.D. Chicago, Ill.	Chicago
1903	W. L. Ballenger, M.D. Chicago, Ill.	C. J. Stillson, M.D. Indianapolis, Ind.	Otto Stein, M.D. Chicago, Ill.	Derrick Vail, M.D. Cincinnati, Ohio	Indianapolis
1904	Edward Jackson, M.D. Denver, Colo.	Dudley Reynolds, M.D. Louisville, Ky.	Otto Stein, M.D. Chicago, Ill.	Derrick Vail, M.D. Cincinnati, Ohio	Denver
1905	Hanau Loeb, M.D. St. Louis, Mo.	Derrick Vail, M.D. Cincinnati, Ohio	Otto Stein, M.D. Chicago, Ill.	George F. Suker, M.D. Chicago, Ill.	Buffalo
1906	Casey Wood, M.D. Chicago, Ill.	J. A. Stucky, M.D. Lexington, Ky.	Otto Stein, M.D. Chicago, Ill.	George F. Suker, M.D. Chicago, Ill.	St. Clair

PAST OFFICERS OF THE ACADEMY

1907—J. A. Stucky, M.D. Lexington, Ky.	R. A. Reeve, M.D. Toronto, Canada	Otto Stein, M.D. Chicago, Ill.	George F. Suker, M.D. Chicago, Ill.	Louisville
1908—Derrick Vail, M.D. Cincinnati, Ohio	Joseph Beck, M.D. Chicago, Ill.	Otto Stein, M.D. Chicago, Ill.	George F. Suker, M.D. Chicago, Ill.	Cleveland
1909—Otto Stein, M.D. Chicago, Ill.	Percy Fridenberg, M.D. New York, N.Y.	S. H. Large, M.D. Cleveland, Ohio	George F. Suker, M.D. Chicago, Ill.	New York
1910—Wendell Reber, M.D. Philadelphia, Pa.	L. B. Lockhard, M.D. Denver, Colo.	S. H. Large, M.D. Cleveland, Ohio	George F. Suker, M.D. Chicago, Ill.	Cincinnati
1911—J. J. Kyle, M.D. Indianapolis, Ind.	F. Parke Lewis, M.D. Buffalo, N. Y.	S. H. Large, M.D. Cleveland, Ohio	George F. Suker, M.D. Chicago, Ill.	Indianapolis
1912—George F. Suker, M.D. Chicago, Ill.	J. W. Murphy, M.D. Cincinnati, Ohio	S. H. Large, M.D. Cleveland, Ohio	L. M. Francis, M.D. Buffalo, N. Y.	Niagara Falls, Ont.
1913—J. W. Murphy, M.D. Cincinnati, Ohio	J. O. McReynolds, M.D. Dallas, Texas	S. H. Large, M.D. Cleveland, Ohio	L. M. Francis, M.D. Buffalo, N. Y.	Chattanooga
1914—J. M. Ray, M.D. Louisville, Ky.	J. M. Ingersoll, M.D. Cleveland, Ohio	S. H. Large, M.D. Cleveland, Ohio	L. M. Francis, M.D. Buffalo, N. Y.	Boston
1915—Joseph Beck, M.D. Chicago, Ill.	Robert S. Lamb, M.D. Washington, D.C.	S. H. Large, M.D. Cleveland, Ohio	L. M. Francis, M.D. Buffalo, N. Y.	Chicago
1916—J. E. Brown, M.D. Columbus, Ohio	T. E. Carmody, M.D. Denver, Colo.	S. H. Large, M.D. Cleveland, Ohio	L. M. Francis, M.D. Buffalo, N. Y.	Memphis
1917—W. L. Dayton, M.D. Lincoln, Nebr.	Edward Stieren, M.D. Pittsburgh, Pa.	S. H. Large, M.D. Cleveland, Ohio	L. M. Francis, M.D. Buffalo, N. Y.	Pittsburgh
1918—Allen Greenwood, M.D. Boston, Mass.	Virginius Dabney, M.D. Washington, D. C.	S. H. Large, M.D. Cleveland, Ohio	L. M. Francis, M.D. Buffalo, N. Y.	Denver

PAST OFFICERS OF THE ACADEMY

1919—J. M. Ingersoll, M.D. Cleveland, Ohio	Melville M. Black, M.D. Denver, Colo.	S. H. Large, M.D. Cleveland, Ohio	Luther C. Peter, M.D. Philadelphia, Pa.	Cleveland
1920—L. M. Francis, M.D. Buffalo, N. Y.	Hal Foster, M.D. Kansas City, Mo.	S. H. Large, M.D. Cleveland, Ohio	Luther C. Peter, M.D. Philadelphia, Pa.	Kansas City
1921—Emil Mayer, M.D. New York, N. Y.	John R. Newcomb, M.D. Indianapolis, Ind.	S. H. Large, M.D. Cleveland, Ohio	Luther C. Peter, M.D. Philadelphia, Pa.	Philadelphia
1922—Walter R. Parker, M.D. Detroit, Mich.	Ross H. Skillern, M.D. Philadelphia, Pa.	S. H. Large, M.D. Cleveland, Ohio	Luther C. Peter, M.D. Philadelphia, Pa.	Minneapolis and St. Paul
1923—T. E. Carmody, M.D. Denver, Colo.	Hunter H. McGuire, M.D. Winchester, Va.	S. H. Large, M.D. Cleveland, Ohio	Luther C. Peter, M.D. Philadelphia, Pa.	Washington, D. C.
1924—W. B. Lancaster, M.D. Boston, Mass.	William P. Wherry, M.D. Omaha, Nebr.	S. H. Large, M.D. Cleveland, Ohio	Luther C. Peter, M.D. Philadelphia, Pa.	Montreal
1925—Horace Newhart, M.D. Minneapolis, Minn.	John M. Wheeler, M.D. New York, N. Y.	S. H. Large, M.D. Cleveland, Ohio	Luther C. Peter, M.D. Philadelphia, Pa.	Chicago
1926—E. C. Ellett, M.D. Memphis, Tenn.	Austin A. Hayden, M.D. Chicago, Ill.	S. H. Large, M.D. Cleveland, Ohio	Luther C. Peter, M.D. Philadelphia, Pa.	Colorado Springs
1927—Ross Hall Skillern, M.D. Philadelphia, Pa.	H. W. Woodruff, M.D. Joliet, Ill.	S. H. Large, M.D. Cleveland, Ohio	William P. Wherry, M.D. Omaha, Nebr.	Detroit
1928—Luther C. Peter, M.D. Philadelphia, Pa.	Frank L. Dennis, M.D. Colorado Springs, Colo.	S. H. Large, M.D. Cleveland, Ohio	William P. Wherry, M.D. Omaha, Nebr.	St. Louis
1929—Harris P. Mosher, M.D. Boston, Mass.	Frank E. Burch, M.D. St. Paul, Minn.	S. H. Large, M.D. Cleveland, Ohio	William P. Wherry, M.D. Omaha, Nebr.	Atlantic City
1930—William H. Wilder, M.D. Chicago, Ill.	Frank R. Spencer, M.D. Boulder, Colo.	S. H. Large, M.D. Cleveland, Ohio	William P. Wherry, M.D. Omaha, Nebr.	Chicago

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1932—S. Hanford McKee, M.D. Montreal, Canada	Gordon B. New, M.D. Rochester, Minn.	William P. Wherry, M.D. Omaha, Nebr.	S. H. Large, M.D. Cleveland, Ohio	Montreal
1933—Burt R. Shurly, M.D. Detroit, Mich.	Nelson Miles Black, M.D. Miami, Fla.	William P. Wherry, M.D. Omaha, Nebr.	S. H. Large, M.D. Cleveland, Ohio	Boston
1934—John M. Wheeler New York, N. Y.	LeRoy A. Schall, M.D. Boston, Mass.	William P. Wherry, M.D. Omaha, Nebr.	S. H. Large, M.D. Cleveland, Ohio	Chicago
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1939—George M. Coates, M.D. Philadelphia, Pa.	William W. Pearson, M.D. Des Moines, Iowa	William P. Wherry, M.D. Omaha, Nebr.	S. H. Large, M.D. Cleveland, Ohio	Chicago
1940—Frank E. Brawley, M.D. Chicago, Ill.	Arthur W. Proetz, M.D. St. Louis. Mo.	William P. Wherry, M.D. Omaha, Nebr.	S. H. Large, M.D. Cleveland, Ohio	Cleveland
1941—Frank R. Spencer, M.D. Boulder, Colo.	Everett L. Goar, M.D. Houston, Texas	William P. Wherry, M.D. Omaha, Nebr.	S. H. Large, M.D. Cleveland, Ohio	Chicago
1942—Ralph I. Lloyd, M.D. Brooklyn, New York	Walter H. Theobald, M.D. Chicago, Ill.	William L. Benedict, M.D. Rochester, Minn.	S. H. Large, M.D. Cleveland, Ohio	Chicago
1943—James A. Babbitt, M.D. Philadelphia, Pa.	Sanford R. Gifford, M.D. Chicago, Ill.	William L. Benedict, M.D. Rochester, Minn.	S. H. Large, M.D. Cleveland, Ohio	Chicago

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American Academy of Ophthalmology and Otolaryngology

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②**Beyer, Louis J.,** '18...449 Delaware av., Buffalo 2, N. Y. **ALR**

Beyer, Theodore E., '31..227 Sixteenth st., Denver 2, Colo. **ALR**

Biehn, J. Telford, '37, Major (RCAMC),
10 Canadian General Hospital, Canadian Army Overseas **OALR**

Bilchick, Edwin B., '35, Major (M.C.),
216 W. 89th, New York 24, N. Y. **ALR**

Billings, Gilbert M., '39
110 S. Sterling, Morganton, N. C. **ALR**

Billings, Robert J., '35...500 Penn av., Pittsburgh 22, Pa. **OP**
Billingsley, J. A., '38, Huron bldg., Kansas City 10, Kans. **OP**
②**Binger, H. E.,** '17, Lowry Med. bldg., St. Paul 2, Minn. **OALR**
②**Binkley, Roy S.,** '17.....Fidelity bldg., Dayton 2, Ohio **OALR**
Birch, John R., '39, Major (M.C.),
Station Hospital, Camp Patrick Henry, Newport News, Va. **ALR**
Birdsall, Clarence H., '37....30 Summer, Haverhill, Mass. **ALR**
Birge, Henry L., '39, Capt. (M.C.),
A.F.A.T.S., Stockton Field, Calif. **OP**
Birsner, L. J., '26, 5344 Englewood pl., St. Louis 21, Mo. **ALR**
Bishop, Frederick J., '36
Medical Arts bldg., Scranton 3, Pa. **ALR**
Bishop, Vernon L., '33..277 Alexander, Rochester 7, N. Y. **ALR**
Bisno, Daniel, '43.....4500 Olive, St. Louis 8, Mo. **OP**
①**Black, Nelson Miles,** '03.Huntington bldg., Miami 32, Fla. **OP**
Black, Paul, '27.....Stuart bldg., Lincoln 8, Nebr. **OALR**
Black, W. Byron, '31.Professional bldg., Kansas City 6, Mo. **ALR**
①**Black, W. D.,** 1900..Metropolitan bldg., St. Louis 3, Mo. **ALR**
②**Blackburn, W. J.,** '19....Reibold bldg., Dayton 2, Ohio **OALR**
Blackmar, Francis B., '31, Maj. (M.C.),
Schick Gen. Hosp., Clinton, Iowa **ALR**
Blair, M. W., '28
N. E. cor. 20th and Chestnut, Philadelphia 3, Pa. **OP**
②**Blair, W. W.,** '17.....Woodland road, Pittsburgh 6, Pa. **OP**
Blaisdell, Irl Hazard, '41
Medical Arts bldg., Syracuse 2, N. Y. **ALR**
Blankfeld, Nathan, '36
429 Lincoln pl., Sta. B, Brooklyn 16, N. Y. **ALR**
Blankstein, Samuel S., '42
606 W. Wisconsin av., Milwaukee 3, Wis. **OP**
Blasdel, Tyler David, '40
110½ S. 18th st., Parsons, Kan. **ALR**
Blassingame, C. D., '23
899 Madison av., Memphis 3, Tenn. **ALR**
Blaydes, J. E., '43
First Nat'l Bank bldg., Bluefield, W. Va. **OP**
①**Bledsoe, R. W.,** '07
First Nat'l Bank bldg., Covington, Ky. **OALR**
Blickenstaff, A. J., '22..Alliance Life bldg., Peoria 2, Ill. **OALR**
Bloch, Charles L., '43, Lt. (M.C.), USNR
Post Dispensary, Quantico, Va. **OP**
Blomberg, Thorsten E., '33.501 Seventh st., Rockford, Ill. **ALR**
Blonder, Edwin J., '34..185 N. Wabash av., Chicago 1, Ill. **ALR**
Bloom, C. Hugh, '32.....65 N. 3rd, Easton, Pa. **ALR**
②**Blue, J. B.,** '16.....62 N. Main, Memphis 3, Tenn. **OALR**
①**Blum, Henry N.,** '14
American Bank bldg., New Orleans 12, La. **OP**
Blumenfeld, Louis, '39
221 Brooklyn av., Brooklyn 13, N. Y. **ALR**
Blumkoltz, Bruno, '43..185 N. Wabash av., Chicago 1, Ill. **ALR**
Boaz, Emmett Daniel, '38
3710 Fifth av., Pittsburgh 13, Pa. **ALR**
Bochner, Maxwell K., '33..329 Bloor st. W., Toronto, Can. **OP**
Bockoven, Sterling, '38
1752 Mass. av. N.W., Washington 6, D. C. **OP**
Boden, Worthey C., '42.....Capt. (M.C.), Knoxville, Iowa **ALR**
Bodenheimer, Ernst, '43.1212 Eutaw pl., Baltimore 17, Md. **OP**
②**Boeckmann, Egil,** '18
Lowry Medical bldg., St. Paul 2, Minn. **OALR**
Boehs, Charles J., '37
Medical Arts bldg., San Antonio 5, Texas **ALR**
Boemer, L. C., '30, Major (M.C.),
3720 Washington blvd., St. Louis 8, Mo. **ALR**
Bogart, Donald Weeks, '40
20 East 53rd st., New York 22, N. Y. **OP**
Boggs, Frank Clinton, '38.....Mills bldg., Topeka, Kan. **OP**
Boice, Ralph E., '35.....316 Michigan, Toledo 2, Ohio **OP**
Boies, Lawrence R., '36
Medical Arts bldg., Minneapolis 2, Minn. **ALR**
①**Boiler, W. F.,** '12, Major (M.C.), 0-197861
603 East 10th st., Rolla, Mo. **OALR**
Boland, William Tilden, '41.....315 Lake, Elmira, N. Y. **OP**
Boles, William M., '41, Capt. (M.C.), 0-333801
A.P.O. 464, c/o PM., New York, N. Y. **OP**
Bolman, Harold Robert, '38
6½ S. Main, Monroeville, Ohio **OP**
Bolotow, Nathan A., '29.126 Waterman, Providence 6, R. I. **ALR**
Bonaccolto, Girolamo, '37
654 Madison av., New York 21, N. Y. **OP**
Bond, Floyd M., '41...625 Broadway, San Diego 1, Calif. **OP**
Bone, Herman D., '20.....19 Pleasant, Gardner, Mass. **OALR**
Bonham, William Lawrence, '39
Medical Arts bldg., Oklahoma City 2, Okla. **ALR**
Bonner, Wm. F., '26
Veterans Administration Facility, Batavia, N. Y. **OALR**
Bonner, William Richard, '42
3 W. White st., Summit Hill, Pa. **OP**
②**Bordley, James Jr.,** '18
4 Charlcote pl., Baltimore 18, Md. **OALR**
Borgeson, E. J., '30
Medical Arts bldg., Minneapolis 2, Minn. **OP**
Borley, William E., '39, Lt. Comdr. (M.C.), USNR, School of Aviation Medicine, Naval Air Sta., Pensacola, Fla. **OP**
Bornstein, Max, '42, Maj. (M.C.), Post Hospital,
1570 S.U., Camp Breckinridge, Ky. **ALR**
Boss, Eugene George, '33..1537 Main, Springfield 3, Mass. **OALR**
Bostwick, D. S., '34.....106 Linwood av., Ardmore, Pa. **ALR**
Boswell, Robert Echo, '41..Fidelity bldg., Dayton 2, Ohio **ALR**
Bothman, Louis, '30...310 S. Michigan av., Chicago 4, Ill. **OP**
②**Boucher, R. B.,** '19
Medical-Dental bldg., Vancouver, B. C., Can. **OALR**
Bourbon, Oliver P., '20
6561 Meridian, Los Angeles 42, Calif. **OALR**
Bourne, Malcolm G., '37...110 W. 3rd, Jamestown, N. Y. **OP**
Bouvy, Harry M., '20
800 Pittock blk., 10th and Washington, Portland, Ore. **OALR**
Bouvy, L. B., '27.....Foley bldg., La Grande, Ore. **OALR**
Bower, Richard L., '43.1103 Grand av., Kansas City 6, Mo. **ALR**
Bowers, Wesley C., '36...17 E. 61st, New York 21, N. Y. **ALR**
①**Boyce, G. H.,** '12
1st Nat'l Bank bldg., Iron Mountain, Mich. **OALR**
Boyce, Wm. A., '31.Roosevelt bldg., Los Angeles 14, Calif. **OP**
Boyd, Charles Wilson, '40, Major (M.C.),
Valley Forge General Hospital, Phoenixville, Pa. **OP**
Boyd, Greydon Gill, '39, Lt. Comdr. (M.C.),
Marine Air Sta., El Toro, Calif. **ALR**
Boyden, Guy Lee, '39.1735 N. Wheeler, Portland 12, Ore. **ALR**
Boyes, T. L., '32....654 Madison av., New York 21, N. Y. **OP**
Bozer, Herrmann E., '23
468 Delaware av., Buffalo 2, N. Y. **ALR**
Bradford, George Edwin, '41, Lt. (M.C.),
Box 24, Navy 121, c/o Fleet Post Office, New York, N. Y. **ALR**
Bradish, Robert F., '42, Col. (M.C.), Port of Embarkation,
Poland and Dauphin sts., New Orleans 12, La. **OP**
Bradley, Jeter C., '34
900 17th st., N.W., Washington 6, D. C. **ALR**
Bradley, John W., '37, Major (M.C.),
76th General Hospital, Vancouver, Wash. **ALR**
Brady, Charles Henry, '43, 205 Ilgenfritz bldg., Sedalia, Mo. **ALR**
Braley, A. E., '39, Lt. Comdr. (M.C.), USNR
U. S. Naval Hospital, St. Albans 12, L. I., N. Y. **OP**
Brandenburg, Kenneth C., '37
110 Pine av., Long Beach 2, Calif. **OALR**

Brandenburg, Nora B., '36
720 N. Michigan av., Chicago 11, Ill. **ALR**

Brandfield, Ira Irving, '40
Capt. (M.C.), Station Hospital, Camp Stewart, Ga. **ALR**

Brandwan, Samuel R., '37, Capt. (M.C.),
Station Hospital, Army Air Base, Richmond, Va. **OP**

Branigan, C. Hugh, '33..114 E. 54th, New York 22, N. Y. **OP**

Brannin, Daniel, '36...Medical Arts bldg., Dallas 1, Texas **OP**

Braswell, James C., '22.Medical Arts bldg., Tulsa 3, Okla. **OALR**

Braunlin, Wm. H., '20
Marion Nat'l Bank bldg., Marion, Ind. **OALR**

①**Brawley, F. E.**, '07...30 N. Michigan av., Chicago 2, Ill. **OP**

Brawner, Leon E., '29..Medical Arts bldg., Atlanta 3, Ga. **ALR**

Bredlau, Ernest A., '29.....2753 W. North, Chicago, Ill. **ALR**

Breeding, Earl G., '32
1801 Eye st. N.W., Washington 6, D. C. **ALR**

Brehmer, Harrison L., '20
First Nat'l Bank bldg., Albuquerque, N. M. **OALR**

Breitstein, M. Lewis, '37
1213 Eutaw pl., Baltimore 17, Md. **ALR**

Bressler, J. L., '34...Medical Arts bldg., Houston 2, Texas **OP**

Breuning, Paul H., '32...133 E. 58th, New York 22, N. Y. **ALR**

Brewer, John D., '20
Baird Brewer Gen. Hosp., Dyersburg, Tenn. **OALR**

Brewster, H. F., '32.913 Richards bldg., New Orleans, La. **ALR**

Briant, Thomas E., '29...28 E. Main, Welland, Ont., Can. **ALR**

③**Bribach, Eugene J.**, '19
Professional bldg., Atchison, Kan. **OALR**

Bricker, Sacks, '31.1101 W. Wyoming, Philadelphia 40, Pa. **ALR**

Brickley, Daniel W., '21
Uhler-Phillips bldg., Marion, Ohio **OALR**

Brickley, Daniel Webster, Jr., '38
Uhler-Phillips bldg., Marion, Ohio **ALR**

Bridgett, Frank Aloysius, '42
3332 Chestnut st., Philadelphia 4, Pa. **ALR**

Briggs, Henry Harrison, Jr., '41, Major (M.C.),
3064 Puritan, Lincoln 2, Nebr. **OP**

Brighton, George R., '36
580 Park av., New York 21, N. Y. **ALR**

Briglia, Frank J., '36, Lt. (M.C.), U.S.N.R.,
819 Wharton, Philadelphia 47, Pa. **ALR**

Brindley, Benjamin, '40, Comdr. (M.C.),
U. S. Naval Hosp., Great Lakes, Ill. **OP**

Brinkerhoff, David E., '39, Lt. Comdr. (M.C.),
320 Professional bldg., Phoenix, Ariz. **ALR**

Bristow, Walter J., '35
1517 Hampton, Columbia 29, S. C. **OALR**

Broadwell, Stuart, Jr., '37...103½ N. 5th, Springfield, Ill. **ALR**

Brobeck, Von Haller, '32
Ferguson bldg., Colorado Springs, Colo. **OP**

①**Brodrick, Frank W.**, '11.....302 First av., Sterling, Ill. **OALR**

Brook, Jeffrey J., '34
231 W. Wisconsin av., Milwaukee 3, Wis. **ALR**

Brooks, Clyde Montgomery, '43, Capt. (M.C.),
Gardiner General Hosp., Chicago 15, Ill. **ALR**

②**Brooks, E. H.**, '15.....Zuelke bldg., Appleton, Wis. **OALR**

Brooks, Earl B., '21....128 N. 13th st., Lincoln 8, Nebr. **OALR**

Brooks, Jack E., '43.....712 Grace, Chicago 13, Ill. **ALR**

Brooks, Morris J., '34...455 Ocean av., Brooklyn 26, N. Y. **ALR**

Brocks, Richmond C. A., '39
1150 Connecticut av., Washington 6, D. C. **ALR**

Broones, Otto, '36.....302 W. 87th, New York 24, N. Y. **ALR**

Brown, Albert L., '26.....Carew Tower, Cincinnati 2, Ohio **OP**

Brown, Andrew George, '42
1010 Huntington bldg., Miami 32, Fla. **OP**

Brown, Audrey O., '27.5057 Woodward av., Detroit 2, Mich. **OALR**

Brown, Charles Lafayette, '38
713-15 Richards bldg., New Orleans 12, La. **ALR**

Brown, Charles Wm., '27
Bank of America bldg., San Diego 1, Calif. **ALR**

Brown, C. J., '34, Captain (M.C.), U.S.N.,
Navy 814, c/c Fleet Post Office, New York, N. Y. **ALR**

①**Brown, Clayton M.**, '12
510 Delaware av., Buffalo 2, N. Y. **ALR**

Brown, Clyde O., '28
3720 Washington blvd., St. Louis 8, Mo. **ALR**

Brown, David Edward, '41
520 Hume-Mansur, Indianapolis 4, Ind. **ALR**

Brown, Earl Henderson, '39...130 S. Scott, Tucson, Ariz. **ALR**

Brown, Henry A., '43.937 Sixth av., S.E., Rochester, Minn. **ALR**

①**Brown, J. M.**, '11...1136 W. 6th, Los Angeles 14, Calif. **ALR**

Brown, John C., '20..Medical Arts bldg., St. Paul 2, Minn. **OALR**

①**Brown, John E.**, '01..370 East Town, Columbus 15, Ohio **OALR**

Brown, Lester A., '39
478 Peachtree st. N.E., Atlanta 3, Ga. **ALR**

Brown, Mortimer G., '21
State Tower bldg., Syracuse 2, N. Y. **ALR**

①**Brown, W. Harold**, '12
Birks bldg., Vancouver, B. C., Can. **OALR**

Brown, Wilbur Max, '43, Lt. Col. (M.C.),
1604 Vincent, Brownwood, Texas **OP**

Brownell, Durwin Hall, '40
Medico-Dental bldg.,San Diego 1, Calif. **ALR**

Brownell, Morton E., '28.106 W. Douglas, Wichita 2, Kan. **OP**

Browning, Andrew J., '35....Mayer bldg., Portland 5, Ore. **OP**

Browning, Birt Lee, '35.....208 Grand, Newburgh, N. Y. **ALR**

Brownsberger, Sidney Burten, '38, Capt. (M.C.),
630 Atkins Drive, Glendale 6, Calif. **OP**

Bruce, G. M., '30, Comdr. (M.C.), U.S.N.R.
F.P.O. San Francisco, Calif. **OP**

①**Bruder, Joseph**, '08....57 W. 57th, New York 19, N. Y. **OALR**

Brumbaugh, John D., '34, Capt. (M.C.), 017351
75 North Wheaton Road, Akron 13, Ohio **OALR**

Brumm, Seth A., '22
Medical Arts bldg.,Philadelphia 3, Pa. **OALR**

Bruner, Abram B., '23..Guardian bldg., Cleveland 14, Ohio **OP**

Bruner, Claude R., '41, Lt. Comdr. (M.C.),
Fleet Post Office, San Francisco, Calif. **OP**

①**Bruner, Wm. E.**, '05..Guardian bldg., Cleveland 14, Ohio **OP**

Brunk, Clifford F., '39
7815 E. Jefferson av., Detroit 14, Mich. **ALR**

Brunner, George Harmon, '40
McCannel Clinic bldg., Minot, N. D. **OP**

③**Brunner, Hans.** '35.307 N. Michigan av., Chicago 1, Ill. **ALR**

Bruno, Joseph M. L., '35...31 Jefferson, Brooklyn 6, N. Y. **OP**

Brusegard, James F., '33.....414 Main, Red Wing, Minn. **OP**

①**Brust, H. O.**, '13..820 S. Crouse av., Syracuse 1, N. Y. **ALR**

Bryan, James H., '43.508 N. Grand blvd., St. Louis 3, Mo. **OP**

Bryan, James L., '28
Bennie-Dillon bldg., Nashville 3, Tenn. **OALR**

Bryan, William T. K., '42
3720 Washington blvd., St. Louis 8, Mo. **ALR**

Bryant, Ben L., '31
2312 Miramar st., Los Angeles 4, Calif. **ALR**

Bryant, Frank L., '37, Major (M.C.), 0237676
P.O. Box 388, Oak Ridge, Tenn. **ALR**

Buchanan, Norman D., '25
129 Murray, Peterborough, Ont., Can. **OALR**

Buchbinder, Murray W., '43, Major (M.C.), Armed
Forces Ind. Sta., 480 Lexington av., New York. N. Y. **ALR**

Bucher, Walter M., '34..4187 Pearl rd., Cleveland 9, Ohio **ALR**

Buck, Clair A., '31, Maj. (M.C.),
191 Glen, Glens Falls, N. Y. **ALR**

Buckingham, Tracy W., '31
405 Broadway, Bismarck, N. D. **ALR**

Buckman, Lewis T., '32..83 S. Franklin, Wilkes-Barre, Pa. **OALR**

Buckman, Samuel T., '40.70 S. Franklin, Wilkes-Barre, Pa. **OALR**

Budetti, Joseph A., '43, Capt. (M.C.),
Oliver General Hospital, Augusta, Ga. **ALR**

Buffington, Wiley R., '35
Hibernia Bank bldg., New Orleans 12, La. **OP**

Buffum, Edward S., '36....40 W. North, Buffalo 2, N. Y. **ALR**

Bullard, Carleton Wm., '32...120 Genesee, Auburn, N. Y. **ALR**

Bullis, John A. E., '39, Lt. (M.C.), U.S.N.R.,
F.P.O., San Francisco, Calif. **OALR**

Bullwinkel, H. G., '31, Comdr. (M.C.),
c/o Fleet P.O., San Francisco, Calif. **ALR**

Bulson, Eugene L., '28..347 W. Berry, Ft. Wayne 2, Ind. **OALR**

Bunker, Paul G., '37.....Newberry bldg., Aberdeen, S. D. **ALR**

Bunnell, Chester William, '35, Lt. Comdr. (M.C.),
9115 193rd st., Hollis, N. Y. **ALR**

Buonaguro, Michael J., '31
589 Lorimer, Brooklyn 11, N. Y. **OP**

Burch, Edward P., '39, Maj. (M.C.), O'Reilly General
Hospital, Springfield, Mo. **OP**

①**Burch, Frank E.**, '07..754 Linwood pl., St. Paul 5, Minn. **OP**

Burchfield, George W., '36....309 Court, Maryville, Tenn. **ALR**

Burdick, Austin F., '25
P. O. Drawer 1378, Lansing 4, Mich. **OALR**

Burgess, John Louis, '40..Medical Arts bldg., Waco, Texas **ALR**

Burgess, Taylor S., '39..Medical Arts bldg., Atlanta 3, Ga. **ALR**

Burgman, James Everette, '38
8 W. Main, Springfield, Ohio **ALR**

Burian, Hermann M., '38..The Eye Clinic, Hanover, N. H. **OP**

Burke, B. Russell, '35, Major (M.C.),
A.P.O. 4777, c/o PM, New York, N. Y. **ALR**

Burke, John R., '27.....207 Elm, Holyoke, Mass. **OALR**

①**Burke, T. A.**, '06.....Rose bldg., Cleveland 15, Ohio **OALR**

Burman, Herman J., '35
1235 Grand Concourse, New York 52, N. Y. **ALR**

Burnham, Howard H., '37
Medical Arts bldg., Toronto 5, Can. **ALR**

Burns, L. J., '30.....1930 Chestnut, Philadelphia 3, Pa. **ALR**

②**Burns, S. S.**, '19.Missouri Theatre bldg., St. Louis 3, Mo. **ALR**

Burritt, N. W., '30....30 Beechwood road, Summit, N. J. **ALR**

Burston, Herschel, '43, Lt. (M.C.),
1917 Wilshire blvd., Los Angeles 5, Calif. **ALR**

Burton, Edwin W., '37.Y.M.C.A. bldg., Charlottesville, Va. **OP**

Bushman, L. B., '27
City National Bank bldg., Omaha 2, Nebr. **OALR**

Butler, Edmund John, '42, 25 Garden st.,
Cambridge 38, Mass. **ALR**

Butler, Harry, '37, Lt. Col. (M.C.),
Sioux Falls Army Air Field, Sioux Falls, So. Dak. **ALR**

Butler, Robert H., '20
112 W. Columbus av., Bellefontaine, Ohio **OALR**

①**Butt, Miriam M.**, '08..2045 Chestnut, Philadelphia 3, Pa. **OP**

Buvinger, Charles W., '21
50 Washington, East Orange, N. J. **OALR**

Buzzard, Josiah F., '31..1110 Thirteenth av., Altoona, Pa. **OALR**

Byrne, Harry Vincent, '38.....301 Essex, Lawrence, Mass. **OP**

Byrne, J. Arthur, '41.....16 Elm, Morristown, N. J. **ALR**

①**Byrnes, Harry F.**, '10..67 Chestnut, Springfield 5, Mass. **OALR**

Byrnes, Victor A., '43, Col. (M.C.),
School of Aviation Medicine, Randolph Field, Texas **OP**

C

Cabot, Clyde Marcus, '38
Medical Arts bldg., Minneapolis 2, Minn. **ALR**

Cady, D. W., '30....65 N. Madison av., Pasadena 1, Calif. **OP**

Cady, F. J., '32.....402 S. Jefferson av., Saginaw, Mich. **OALR**

Cahill, James F., '37.....428 S. Salina, Syracuse 2, N. Y. **OP**

Calcote, Royal J., '36, Comdr. (M.C.),
c/o Fleet Post Office, San Francisco, Calif. **OP**

①**Caldwell, Robert**, '12
New Donaghey bldg., Little Rock, Ark. **OALR**

①**Calhoun, F. Phinizy**, '08
478 Peachtree st. N.E., Atlanta 3, Ga. **OP**

Callahan, Alston, '38, Lt. (M.C.),
RFD 4, Box 26x, Albany, Ga. **OP**

Calvert, Raymond R., '36, Maj. (M.C.),
Station Hosp., Selfridge Field, Mich. **OP**

Cammisa, James J. Vincent, '43, Comdr. (M.C.),
U. S. Naval Air Station, Med. Dept., Quonset Pt., R. I. **OALR**

Camp, W. E., '20.Medical Arts bldg., Minneapolis 2, Minn. **OALR**

Campbell, A. A., '30.....86 Bloor st. W., Toronto, Can. **ALR**

②**Campbell, C. H.**, '18.2514½ Thirteenth, Columbus, Nebr. **OALR**

②**Campbell, Duncan A.**, '19
David Whitney bldg., Detroit 26, Mich. **OALR**

Campbell, Edward H., '38, Lt. Comdr. (M.C.), USNR
Naval Hospital, Norfolk, Va. **ALR**

Campbell, Glenn S., '37, Capt. (M.C.), U. S. Naval Med.
Supply Depot, Pearl and Sands sts., Brooklyn, N. Y. **ALR**

Campbell, Mac D., '27, Capt. (M.C.),
Station Hospital, A.A.F.S.A.T., Orlando, Fla. **OALR**

Campbell, Paul A., '39, Lt. Col. (M.C.),
School of Aviation Med., Randolph Field, Texas **ALR**

Campbell, Sherburne, '36, Lt. Comdr. (M.C.),
U. S. Naval Hosp., Philadelphia 45, Pa. **OP**

③**Campbell, W. K.**, '19..96 Third av., Long Branch, N. J. **OALR**

Campbell, William, '39.144 S. Harrison, E. Orange, N. J. **ALR**

Campbell, Wm. E., Jr., '36
Medical Arts bldg., Atlanta 3, Ga. **OP**

Canfield, Norton, '33, Lt. Col. (M.C.),
789 Howard av., New Haven 4, Conn. **ALR**

Cannon, Harold J., '43
536 W. Wisconsin av., Milwaukee 3, Wis. **ALR**

Capeles, Thomas Francis, '42
191 Merrimack st., Haverhill, Mass. **ALR**

Cardwell, Edgar P., '29, Major (M.C.),
B. T. C. No. 6, Lincoln Field, Lincoln, Nebr. **ALR**

Carelli, Paul Vincent, '41
3626 W. Chicago av., Chicago, 51, Ill. **OP**

Carl, Evan Elsworth, '40
606 W. Wisconsin av., Milwaukee 3, Wis. **OP**

Carmody, Raymond F., '37.....504 Broadway, Gary, Ind. **OP**

①**Carmody, T. E.**, '04
Metropolitan bldg., Denver 2, Colo. **ALR-PI S**

Caron, Armand L., '39....36 Pleasant, Worcester 8, Mass. **ALR**

Carpenter, Chapin, '30..1930 Chestnut, Philadelphia 3, Pa. **OP**

Carrasquillo, Honorio Fernando, '41
P. O. Box 128, Utuado, Puerto Rico **OP**

Carroll, Frank D., '37.635 W. 165th, New York, 32, N. Y. **OP**
Also: Blind Brook Lodge, Rye, N. Y.

Carroll, Walter J. E., '37..5 Chestnut, Arlington 74, Mass. **ALR**

Carruth, Howard E., '28
Medical Arts bldg., Portland 5, Ore. **OALR**

②**Carson, W. E.**, '17....Jenkins Arcade, Pittsburgh 22, Pa. **OP**

Carter, Albert A., '37.....419 Boylston, Boston 16, Mass. **OP**

Carter, E. D., '30.....Central bldg., Wichita 2, Kan. **ALR**

Carter, John M., '29.David Whitney bldg., Detroit 26, Mich. **ALR**

Carter, Leland F., '37
David Whitney bldg., Detroit 26, Mich. **OP**

①Carter, W. W., '08.....6 Chestnut av., Bronxville, N. Y. **ALR**

Caruthers, Samuel B., '37..Grenada Clinic, Grenada, Miss. **OALR**

①Cary, E. H., '12.....Medical Arts bldg., Dallas 1, Texas **OALR**

②Cary, G. C., '18.....Canon Block, Grand Junction, Colo. **OALR**

Case, Paul Henry, '39.....15 E. Monroe, Phoenix, Ariz. **OP**

Cassady, J. Vernal, '28.Sherland bldg., South Bend 9, Ind. **OALR**

Cassidy, Waldron A., '31
Medical Arts bldg., Omaha 2, Nebr. **ALR**

Casten, Virgil G., '32, Lt. Comdr. (M.C.),
520 Commonwealth av., Boston 15, Mass. **OP**

Castroviejo, Ramon, '31
635 W. 165th, New York 32, N. Y. **OP-PI S**

Cave, Linus S., '29...State Tower bldg., Syracuse 2, N. Y. **ALR**

Cawthorne, Harold Jesse, '41
239 Pipestone, Benton Harbor, Mich. **ALR**

Cecha, Albin H., '35, c/o Mrs. Nancy Cecha,
4437 Woolworth av., Omaha 5, Nebr. **OP**

Cetner, John A., '43, Lt. (M.C.), U.S. Navy Flight
Preparatory School, Williams College, Williamstown, Mass. **OP**

Chadwick, Ira B., '20.....717½ Walnut, Coffeyville, Kan. **OALR**

Chaffers, Wm. H., '31.....190 Bates, Lewiston, Maine **ALR**

Chainski, Edward L., '32.....130 N. Wells, Chicago 6, Ill. **ALR**

Chamberlain, Webb P., '31
7405 Detroit av., Cleveland 2, Ohio **OP**

Champion, Albert N., '36
Medical Arts bldg., San Antonio 5, Texas **ALR**

Chandler, Arthur C., '42, Major (M.C.), 0-259313
A.P.O. 629, c/o P.M., New York N. Y. **OP**

Chandler, G. E., '30.....Huntington bldg., Miami 32, Fla. **ALR**

Chandler, Paul Austin, '38
5 Bay State Road, Boston 15, Mass. **OP**

Chapman, E. J., '43...331 Haywood bldg., Asheville, N. C. **ALR**

Chapman, Katharine H., '36
304 Burns bldg., Colorado Springs, Colo. **OP**

Chapman, S. Jefferson, '26
Burns bldg., Colorado Springs, Colo. **ALR**

②Chapman, Vernon A., '15
435 N. Palm Canyon drive, Palm Springs, Calif.,
and 324 E. Wisconsin av., Milwaukee 2, Wis. **OALR**

Charles, Joseph W., '22...Humboldt bldg., St. Louis 3, Mo. **OP**

②Charlton, C. Coulter, '16
124 S. Illinois av., Atlantic City, N. J. **ALR**

Chase, Carl Clarence, '39....121 Main, Middletown, Conn. **OP**

②Chase, E. Frank, '18.....Cobb bldg., Seattle 1, Wash. **OALR**

Chase, S. B., '25.....Carver bldg., Ft. Dodge, Iowa **OALR**

Chase, W. D., '24.....230 E. Broad, Bethlehem, Pa. **ALR**

Cheek, Carey, '38.....Medical Arts bldg., Springfield, Mo. **OP**

①Chenery, W. E., 377 Commonwealth av., Boston 15, Mass. **ALR**

Cheney, James W., '20...Schweiter bldg., Wichita 2, Kan. **OALR**

Cheney, Ralph E., '36, Major (M.C.),
Station Hosital, Camp Stoneman, Calif. **OP**

Chenoweth, Charlie Everette, '42
Forester's bldg., Mason City, Iowa **ALR**

Childers, Robert J., '29.....604 Park av., Plainfield, N. J. **ALR**

②Childs, H. A., '15.....1016 W. Adams, Creston, Iowa **OALR**

Chipman, L. DeV., '25...42 Coburg, St. John, N. B., Can. **OALR**

Chisholm, Julian F., Jr., '36
201 Devonshire st., Boston 10, Mass. **OP**

Christoph, Carl H., '28.30 N. Michigan av., Chicago 2, Ill. **OALR**

Cinelli, Albert A., '36..1021 Park av., New York 28, N. Y. **ALR**

Clapp, C. A., '43.....513 N. Charles, Baltimore 1, Md. **OP**

Clark, Cecil P., '31.922 Hume-Mansur, Indianapolis 4, Ind. **OP**

②Clark, H. S., '16....2615 Park av., Minneapolis 7, Minn. **OALR**

Clark, Howard Elton, '39, 576 Hartnell st., Monterey, Calif. **OALR**

Clark, I. G., '20.....188 E. State, Columbus 15, Ohio **OALR**

①Clark, J. Sheldon, '14...State Bank bldg., Freeport, Ill. **OALR**

Clark, James W., '37, Major (M.C.),
3247 Louisville, El Paso, Texas **OP**

②Clark, L. H., '15....337 Monroe av., Rochester 7, N. Y. **OALR**

Clark, Willard C., '43..530 Fidelity bldg., Dayton 2, Ohio **OP**

Clark, William B., '34
American Bank bldg., New Orleans 12, La. **OP**

Clarke, Clement C., '39, Major (M.C.), Sta. Hosp.,
MacDill Field, Tampa 8, Fla. **OP**

Clarke, George L., '38...910 Chalmers, Detroit 15, Mich. **ALR**

Clarke, Samuel Tracy, '41
240 West First Street, Reno, Nev. **OP**

②Clay, Grady E., '17....Medical Arts bldg., Atlanta 3, Ga. **OP**

Clayton, Milton Burns, '42
Southern R. R. Office bldg., Washington 13, D. C. **ALR**

Cleary, James Augustine, '40, Maj. (M.C.),
1580th Service Unit, Camp Campbell, Ky. **ALR**

Cleff, Oscar, '26.....55 E. Washington, Chicago 2, Ill. **OALR**

②Clement, C. C., '18...30 N. Michigan av., Chicago 2, Ill. **OALR**

Clement, L. O., '30.....Guardian bldg., Salem, Ore. **OALR**

Clerf, Louis H., '23.....1530 Locust, Philadelphia 2, Pa. **ALR**

Clifford, T. P., '27.David Whitney bldg., Detroit 26, Mich. **OALR**

Cline, Samuel, '32.....416 Marlborough, Boston 15, Mass. **ALR**

①Clothier, J. V., '06.....Kane bldg., Pocatello, Idaho **OALR**

Clothier, William L., '43
407-11 Kane bldg., Pocatello, Idaho **OP**

Coachman, Edwin H., '39
Manhattan bldg., Muskogee, Okla. **ALR**

Coakley, Leo P., '41...Medical Arts bldg., Omaha 2, Nebr. **ALR**

Coates, George M., '21.....1721 Pine, Philadelphia 3, Pa. **ALR**

①Cobb, Edwin, '12.1680 N. Vine st., Los Angeles 28, Calif. **OALR**

Cody, Claude C., Jr., '28
1304 Walker av., Houston 2, Texas **ALR**

Cogan, David G., '39.....30 Clark, Belmont, Mass. **OP**

Cohen, Bernard M., '38, Lt. (M.C.), USNR
Navy 151, c/o Fleet P.O., San Francisco, Calif. **ALR**

Cohen, Herman B., '27...255 S. 17th, Philadelphia 3, Pa. **ALR**

Cohen, Jerome H., '42, Capt. (M.C.),
Station Hospital B.T.C. 10, Greensboro, N. C. **OP**

Cohen, Louis B., '36....1320 Arrott, Philadelphia 24, Pa. **OP**

①Cohen, Martin, '10.....29 E. 64th, New York 21, N. Y. **OP**

Cohen, Samuel, '21....1920 Pine, Philadelphia 3, Pa. **ALR-PI S**

Cohoe, Don A., '30.13535 Woodward av., Detroit 3, Mich. **ALR**

Cole, Nathan B., '35, Lt. Col. (M.C.),
104 Market, Perth Amboy, N. J. **OALR**

Coleman, Benjamin, '41, Capt. (M.C.),
Battey General Hospital, Rome, Ga. **ALR**

Coleman, Joseph Arvine, '40, Capt. (M.C.),
Station Hospital, Fort Hancock, N. J. **ALR**

Coleman, Lester Laudy, '41
162 E. 80th, New York 21, N. Y. **ALR**

Collins, Braswell E., '42, Major (M.C.),
Station Hospital, Hunter Field, Ga. **ALR**

①Collins, E. W., '10.....1578 Humboldt, Denver 6, Colo. **ALR**

①Collins, J. D., '14.....187 Main, Northampton, Mass. **ALR**

Collins, James T., '40...Republic bldg., Cleveland 15, Ohio **ALR**

①Colver, B. N., '13.436 S. Boyle av., Los Angeles 33, Calif. **ALR**

Combacker, Leon C., '35
101½ Lincoln av., Fergus Falls, Minn. **OP**

Combs, Arnold Brefoe, '42, Lt. (M.C.), U.S.N.R.,
Camp Peary, Williamsburg, Va. **ALR**

Combs, Fielding, '42.Nissen bldg., Winston Salem 3, N. C. **ALR**

Comee, W. C., '22, 6230 N. Kenmore av., Chicago 20, Ill. **OALR**

Conboy, John E., '35
2265 University av., New York 53, N. Y. **OP**

Cone, Alfred J., '29, Major (M.C.),
Station Hospital A.A.F.T.S., Ellington Field, Texas **ALR**
Conley, John J., '43, Capt. (M.C.),
Tilton General Hospital, Fort Dix, N. J. **ALR**
Connell, Evan Shelby, '40
Commerce bldg., Kansas City 6, Mo. **OP**
Connelly, Edgar J., '42.....Roshek bldg., Dubuqe, Iowa **ALR**
Connole, J. V., '32
Miners Nat'l Bank bldg., Wilkes-Barre, Pa. **OP**
Connor, Arthur B., '42..Sweet bldg., Fort Lauderdale, Fla. **ALR**
Connor, C. E., '20...Lowry Med. bldg., St. Paul 2, Minn. **ALR**
Connor, Joseph Joyce, '42....750 Main, Hartford 3, Conn. **OP**
Constans, George M., '29....Clinic bldg., Bismarck, N. D. **OP**
Constantine, Elizabeth Frederick, '43
340 E. 63rd, New York, 21, N. Y. **OP**
Constantine, K. W., '36
2820 Berwick Road, Birmingham 5, Ala. **OP**
Constantinople, P. S., '29
1801 Eye St. N.W., Washington 6, D. C. **ALR**
Conty, Anthony J., '37
318 Forty-eighth st., Union City, N. J. **ALR**
Conway, J. M., '30....Jenkins Arcade, Pittsburgh 22, Pa. **ALR**
Conway, William S., '35, Capt. (M.C.), Halloran
General Hospital, Staten Island, N. Y. **OALR**
Cook, Raymond C., '36, Lt. Comdr. (M.C.), U.S.N.A.S.
Dispensary, Pensacola, Fla. **OP**
Cook, W. Albert, '24....Medical Arts bldg., Tulsa 3, Okla. **OALR**
Cooley, Carl C., '37....Medical Arts bldg., Norfolk 10, Va. **ALR**
Coombs, A. J., '32....30 N. Michigan av., Chicago 2, Ill. **ALR**
Coon, Eugene H., '35...131 Fulton av., Hempstead, N. Y. **ALR**
Cooper, Edmond L., '39
414 David Whitney bldg., Detroit 26, Mich. **OP**
Cooper, F. Maxey, '41
Medical Arts bldg., Oklahoma City 2, Okla. **OP**
Cooper, James H., '43, 316 Michigan st., Toledo 2, Ohio **OP**
Cooper, Kemp G., '39..Metropolitan bldg., Denver 2, Colo. **ALR**
Cooper, Roy C., '32....510 S. Aiken av., Pittsburgh 6, Pa. **ALR**
Cooperman, Harold O., '37, Suite 210, Beverly Professional
bldg., 9615 Brighton Way, Beverly Hills, Calif. **OP**
Copps, Lyman A., '23..Marshfield Clinic, Marshfield, Wis. **OALR**
Corboy, Philip M., '36 Comdr. (M.C.), Navy 128,
Box 1394, c/o Fleet Post Office, San Francisco Calif. **OP**
Cordes, Frederick C., '33..384 Post, San Francisco 8, Calif. **OP**
Cordray, David P., '41, Lt. Comdr. (M.C.), U.S.N.R.,
c/o Fleet Post Office, San Francisco, Calif. **ALR**
Cornelius, C. E., '40, Lt. Comdr. (M.C.),
U. S. Naval Hospital, Shoemaker, Calif. **ALR**
①**Corser, John B.**, '11...415 Mulberry st., Scranton 3, Pa. **OP**
Cortopassi, A. J., '30
324 S. Washington av., Saginaw, Mich. **ALR**
Cortopassi, Vital Elwin, '41, Capt. (M.C.),
A.P.O. 837, c/o Postmaster, New Orleans, La. **ALR**
Corya, H. W., '30.....30 E. 40th, New York 16, N. Y. **ALR**
Cosgrove, Kingsley White, '40
115 E. Capitol, Little Rock, Ark. **OALR**
Costen, J. B., '25.Beaumont Medical bldg., St. Louis 8, Mo. **ALR**
Costenbader, Frank Duncan, '40
1150 Connecticut av., Washington 6, D. C. **OP**
Coston, Tullos O., '40
Medical Arts bldg., Oklahoma City 2, Okla. **OP**
②**Cott, C. C.**, '15.....333 Linwood av., Buffalo 9, N. Y. **ALR**
Cotten, Willis Frank, '43.....202 State, McComb, Miss. **ALR**
Cottle, Maurice H., '36.30 N. Michigan av., Chicago 2, Ill. **ALR**
Courtney, Robert H., '35
Professional bldg., Richmond 19, Va. **OP**
Cowan, Alfred, '28.....1930 Chestnut, Philadelphia 3, Pa. **OP**
Cowan, Clyde R., '33
69 Bay State road, Back Bay, Boston 15, Mass. **ALR**
Cowan, Thomas H., '41, Capt. (M.C.),
Sta. Hosp., Camp Myles Standish, Taunton, Mass. **OP**
Cowen, Jack P., '37, Lt. (M.C.),
U. S. N. Medical Supply Depot, Brooklyn 1, N. Y. **OP**
②**Cowper, H. W.**, '19.....543 Franklin, Buffalo 2, N. Y. **OP**
Cox, Charles L., '36
Pere Marquette bldg., New Orleans 12, La. **ALR**
Cox, Kelly, '39.....Medical Arts bldg., Dallas 1, Texas **OP**
Cox, Ronald A., '37
1779 Mass. av., N.W., Washington 6, D. C. **OP**
Cracovaner, Arthur J., '35, Major (M.C.),
AAF Regional Sta. Hosp., Coral Gables, Fla. **ALR**
Craddock, William Henry, '38, Lt. Col. (M.C.),
Patterson Field, Fairfield, Ohio **ALR**
Craft, Kenneth L., '20
1002 Hume-Mansur bldg., Indianapolis 4, Ind. **ALR**
Crage, Francis M., '35..25 E. Washington, Chicago 2, Ill. **OP**
Craig, Paul C., '33.....232 N. 5th, Reading, Pa. **OP**
Cramton, Edward A., '37....33 Malin, St. Johnsbury, Vt. **OALR**
Crane, Cyril Vincent, '38, Major (M.C.),
Crane, Cyril Vincent, '38, Major (M.C.), address unknown **OP**
Crane, W. R., '29
Room 1026, 727 W. 7th, Los Angeles 14, Calif. **ALR**
Cranston, William J., Jr., '43, Capt. (M.C.),
Station Hospital, S.A.A.C.C., San Antonio, Texas **OP**
Crawford, H. E., '43..139 Kinoole St., Hilo, Hawaii, T. H. **OP**
Creasy, Raymond C., '37..67 E. 59th, New York 22, N. Y. **ALR**
Crebbin, Alexander R., '38
Maison Blanche bldg., New Orleans 16, La. **OP**
Cregar, John S., '39.....150 Harrison, East Orange, N. J. **OP**
①**Creighton, Wm. J.**, '10..37 S. 20th, Philadelphia 3, Pa. **OP**
Crespo, Jose E., '41.....28 W. 95th, New York 25, N. Y. **ALR**
Cress, William J., '34.....318 W. Market, Pottsville, Pa. **ALR**
Creveling, Earle LaMont, '42
17 No. Virginia st., Reno, Nev. **ALR**
①**Crisp, William H.**, '13, Metropolitan bldg., Denver 2, Colo. **OP**
Criswell, Robert H., '37....Phoenix bldg., Bay City, Mich. **OP**
②**Crockett, R. L.**, '17.....112 Farrier av., Oneida, N. Y. **OP**
Croissant, Charles A., '31..390 Main, Worcester 8, Mass. **ALR**
Croll, Leo J., '39, Capt. (M.C.), Ashford General Hosp.,
White Sulphur Springs, W. Va. **ALR**
Croman, Joseph M., Jr., '24
115 Cass av., Mt. Clemens, Mich. **OALR**
①**Cross, A. E.**, '14.....390 Main, Worcester 8, Mass. **OP**
②**Cross, G. H.**, '19.....525 Welsh, Chester, Pa. **OP**
Crossen, Henry F., '39
David Whitney bldg., Detroit 26, Mich. **ALR**
①**Crossley, E. R.**, '11..6821 N.W. 4th av., Miami 38, Fla. **OALR**
Croushore, James E., '35, Major (M.C.),
2045 Edison, Detroit 6, Mich. **ALR**
Crume, John J., '31.....724 Polk, Amarillo, Texas **ALR**
Crumrine, Norman R., '28.....595 Third st., Beaver, Pa. **OALR**
Cruthirds, Archie Edward, '40
Professional bldg., Phoenix, Ariz. **ALR**
①**Culbertson, L. R.**, '98..Masonic Temple, Zanesville, Ohio **OALR**
Culler, Arthur M., '32, Comdr. (M.C.),
1007 Baldwin av., Norfolk, Va. **OP**
①**Cullom, M. M.**, '10
Bennie-Dillon bldg., Nashville 3, Tenn. **OALR**
Culpepper, William S., '32, Col. (M.C.),
Station Hospital, Camp Stoneman, Calif. **ALR**
Cummings, Edward J., '37
1835 Eye St. N.W., Washington 6, D. C. **ALR**
Cummings, George O., '30...47 Deering, Portland 4, Maine **ALR**

Cunning, Daniel S., '28..121 E. 60th, New York 22, N. Y. OALR
Cunningham, Bernard P., '43, Capt. (M.C.),
Army-Navy Gen. Hosp., Hot Springs, Ark. ALR
Cunningham, Henry Kelso, '42
Medical Arts bldg., Knoxville 1, Tenn. ALR
Cunningham, John C., '43, Capt. (M.C.),
Patterson Field, Fairfield, Ohio OP
②Curdy, R. J., '18....1102 Grand av., Kansas City 6, Mo. OP
Curran, Desmond, '37, Lt. (M.C.),
Federal Res. Bank bldg., Kansas City 6, Mo. OP
②Curran, E. J., '19
Federal Res. Bank bldg., Kansas City 6, Mo. OP
Curran, Kevin, '39
Federal Res. Bank bldg., Kansas City 6, Mo. OP
①Curry, Glendon E., '12
Diamond Bank bldg., Pittsburgh 22, Pa. OP
Curtin, Eugene A., '23.....506 Spruce, Scranton 3, Pa. OALR
Curtin, John F., '36
Medical Arts bldg., Minneapolis 2, Minn. ALR
Cushman, Beulah, '24....25 E. Washington, Chicago 2, Ill. OP
Cusick, Paul L., '43, Major (M.C.),
Percy Jones Gen. Hosp., Battle Creek, Mich. OP
Cutino, R.M., '30.....79 Pierrepont, Brooklyn 2, N. Y. OP
Cutler, Abraham Maurice, '41
750 Ocean av., Brooklyn 26, N. Y. ALR
①Cutler, Franklin E., '12.....Wecoma, Ore. ALR
Cutler, Harold Masha, '42, Capt. (M.C.), 0-436867,
A.P.O. 795, c/o Postmaster, New York, N. Y. ALR
Cutler, Norman Leon, '38, Lt. Col. (M.C.),
Veterans Administration, Jackson, Miss. OP

D

Dahl, Elmer O., '33
Medical Arts bldg., Minneapolis 2, Minn. ALR
Dailey, Gilbert L., '27.....618 N. 3rd, Harrisburg, Pa. OALR
Daily, Louis, '26....Medical Arts bldg., Houston 2, Texas OALR
Daily, Ray K., '26...Medical Arts bldg., Houston 2, Texas OALR
Daisey, Willard O., '37.....Fruitland, Md. ALR
Daley, Jacob, '41.....104 E. 40th, New York 16, N. Y. ALR
Dalton, S. Eugene, '38
101 S. Surrey Pl., Ventnor City, N. J. ALR
Dame, Lawrence R., '42, Maj. (M.C.), 0445152,
A.P.O. 923, c/o Postmaster, San Francisco, Calif. OP
Damitz, John Cyril, '43, Lt. (M.C.), USNR, Box 45,
Navy 121, Fleet Post Office, New York, N. Y. OALR
Daniel, Ruby K., '43...Medical Arts bldg., Dallas 1, Texas OP
Danielson, Ralph W., '34
Metropolitan bldg., Denver 2, Colo. OP
Darmer, George A., '21.....Terminal bldg., Aurora, Ill. OALR
Darrough, L. E., '32, Comdr. (M.C.),
Naval Air Station, San Diego, Calif. ALR
Darrow, C. Howard, '28
3020 E. Colorado st., Pasadena 8, Calif. ALR
Dart, Merrill Oren, '40, Maj. (M.C.)
A.A.F. Redistribution Sta. No. 2, Miami Beach, Fla. ALR
Davidson, H. P., '24...30 N. Michigan av., Chicago 2, Ill. OP
Davidson, Morris, '29.....16 Park av., New York 16, N. Y. OP
Davies, H. F., '30.1835 Eye st. N.W., Washington 6, D. C. ALR
Davies, Morgan C., '32...269 E. State, Columbus 15, Ohio OP
①Davies, T. Alexander, '13.578 Sherbourne, Toronto, Can. OALR
Davies, Windsor S., '36, U. S. Naval Hospital,
Treasure Island, San Francisco, Calif. OP
Davis, Arthur Henry, '41.Medical Arts bldg., Tulsa 3, Okla. ALR
Davis, David, '34.1835 Eye st. N.W., Washington 6, D. C. ALR
Davis, Frederick Allison, '27
224 W. Washington av., Madison 3, Wis. OP
②Davis, George E., '15..14 E. 90th, New York 28, N. Y. OALR
Davis, Hugh P., '37, Maj. (M.C.), 0-466967,
A.P.O. 758, c/o Postmaster, New York, N. Y. ALR
Davis, James Edward, '40.179 Allyn st., Hartford 3, Conn. ALR
Davis, John Calvin, Jr., '28..Aquila Court, Omaha 2, Nebr. OALR
Davis, Leo Lloyd, '42
1437 Monaco Parkway, Denver 7, Colo. OP
Davis, Oscar, '36, Capt. (M.C.), USN,
U. S. Naval Hospital, Norman, Okla. OALR
Davis, Ralph F., '20..Medical Arts bldg., Portland 5, Ore. OALR
Davis, T. Carroll, '31..3128 N. Broad, Philadelphia 32, Pa. ALR
Davis, Thomas W., '23
O'Hanlon bldg., Winston-Salem 3, N. C. OALR
Davis, Warren B., '34
135 S. 18th, Philadelphia 3, Pa. ALR-PI S
Davis, Wm. C., '30.....150 E. Broad, Columbus 15, Ohio OP
②Davis, Wm. Thornwall, '19
927 Seventeenth st. N.W., Washington 6, D. C. OP
Davison, Francis W., '36....Geisinger Hosp., Danville, Pa. ALR
Day, Kenneth M., '30.121 University pl., Pittsburgh 13, Pa. ALR
②Dayton, G. O., '18.1401 S. Hope, Los Angeles 15, Calif. OALR
Deal, John F., '28.....522 E. Capitol av., Springfield, Ill. OALR
Dean, Alfred, '23
Medical Arts bldg., Grand Rapids 2, Mich. OP
①Dean, F. W., '03.....22 S. 6th, Council Bluffs, Iowa OALR
Dean, L. W., Jr., '38
3720 Washington blvd., St. Louis 8, Mo. ALR
②Dean, Walter, '17..300 Francis bldg., Louisville 2, Ky. OALR
Dearmin, Robert M., '31, Major (M.C.), Station Hospital,
Westover Field, Chicopee Falls, Mass. ALR
De Blois, Elizabeth, '39
395 Commonwealth av., Boston 15, Mass. ALR
DeBoe, M. P., '20.....Huntington bldg., Miami 32, Fla. OALR
②Decker, J. C., '18.....Frances bldg., Sioux City 8, Iowa OALR
Decker, Phillip Harold, '42
25 W. Third st., Williamsport 5, Pa. ALR
Decker, Russell M., '38.65 N. Madison, Pasadena 1, Calif. ALR
Deems, Myers B., '43, Major (M.C.),
Fitzsimons Gen. Hosp., Box 6087, Denver 8, Colo. ALR
Deeton, William L., '38
13125 Shaker Square, Cleveland 20, Ohio ALR
Defnet, William A., '21.28 Adams av. W., Detroit 26, Mich. ALR
Deichler, L. Waller, '20.1930 Chestnut, Philadelphia 3, Pa. OALR
Deitchman, Louis S., '39, Major (M.C.),
182nd. Gen. Hosp., LaGarde Gen. Hosp., New Orleans, La. ALR
Delancy, Adrian Joseph, '41, Comdr. (M.C.),
1519 31st st. N. W., Washington, D.C. ALR
Delaney, James H., '31.....138 W. 9th, Erie, Pa. OP
Delman, Abraham H., '39, Maj. (M.C.), Veterans Adm.
Hosp., 130 W. Kingsbridge rd., Bronx 63, N. Y. ALR
DeLozier, Leonard Cecil, '41, Lt. (M.C.),
U. S. Naval Training Sta., Disp. 109, Great Lakes, Ill. ALR
Delph, John F., '28..700 N. Michigan av., Chicago 11, Ill. ALR
Denison, Ward C., '31, Comdr. (M.C.), U. S. Navy No. 138,
c/o Fleet P. O., New York, N. Y. ALR
Denman, Francis E., '38
541 Lincoln road, Miami Beach, Fla. ALR
①Dennis, F. L., '06..2745 Barnson pl., San Diego 3, Calif. ALR
①Dennis, George J., '10..25 E. Washington, Chicago 2, Ill. ALR
Denyes, Gerald Foster, '40...316 Michigan, Toledo 2, Ohio ALR
Depping, C. W., '30.....17 W. 54th, New York 19, N. Y. ALR
Dessoff, Joseph, '38
1726 Eye st. N.W., Washington 6, D. C. OP
De Stio, Daniel S., '39..Highland bldg., Pittsburgh 6, Pa. ALR
Deutsch, William, Jr., '42, Major (M.C.),
1001 W. Gilbert, Muncie, Ind. ALR

DeVoe, Arthur Gerard, '41, Capt. (M.C.),
Halloran General Hospital, Staten Island, N. Y. **OP**
De Vries, Harold G., '38.....36 E. 8th, Holland, Mich. **ALR**
Dewar, Murray M., '32
Medical Arts bldg., Grand Rapids 2, Mich. **OP**
DeWeese, Clarence, '20
201 W. Second st., Lexington 15, Ky. **OALR**
①**Dewey, C. H.,** '12, Lt. Col. (M.C.), U.S.A.,
90 Church, New York 7, N. Y. **OALR**
DeWitt, Edward N., '28.881 Lafayette, Bridgeport 4, Conn. **OP**
DeWolf, Charles W., '38.....8 Porter st., Melrose, Mass. **ALR**
Dexter, Milton A., '30.....416 Georgia, Vallejo, Calif. **ALR**
Dezer, Charles N., Jr., '41....210 Main, Hackensack, N. J. **ALR**
Dickerson, William E., '35, Major (M.C.),
802 Masonic Temple, Danville, Va. **ALR**
Dickinson, Wm. G., '36....State Tower, Syracuse 2, N. Y. **OP**
Dickson, J. Charles, '31
Medical Arts bldg., Houston 2, Texas **ALR**
Dickson, Robert L., '38...4803 Penn, Philadelphia 24, Pa. **ALR**
Diess, Robert G., '42, Major (M.C.),
Oliver Gen. Hosp., Forest Hills, Augusta, Ga. **OP**
Dietl, Ernest L., '40, Capt. (M.C.), 0-333476,
APO 647, c/o Postmaster, New York, N. Y. **ALR**
Dill, J. Lewis, '38..Henry Ford Hospital, Detroit 2, Mich. **ALR**
Dillemuth, Frederick Joseph, '41
404 E. 154th, New York 55, N. Y. **ALR**
Dillon, Elmo E., '38.....1952 Ridge rd., Homewood, Ill. **OALR**
Dimmitt, Frank W., '34.78 Waterman, Providence 6, R. I. **OALR**
Dimond, Waldo B., '33, Major (M.C.), 0-227956,
A.P.O. 9826, c/o Postmaster, New York, N. Y. **ALR**
Dinan, Thomas Edward, '41, Lt. Comdr. (M.C.),
44 Beechwood Dr., Bayridge, E. Greenwich, R. I. **ALR**
Dintenfass, Henry, '22....1305 Spruce, Philadelphia 7, Pa. **ALR**
Diven, Wilbur Lester, '41...406½ Main, Bismarck, N. D. **ALR**
Dix, Harold C., '42...405 N. Charles st., Baltimore 1, Md. **OP**
Dixon, Fred W., '29.....Rose bldg., Cleveland 15, Ohio **ALR**
①**Dixon, G. S.,** '09....255 W. 108th, New York 25, N. Y. **R**
Dixon, O. Jason, '28.Professional bldg., Kansas City 6, Mo. **ALR**
①**Dixon, W. E.,** '18.....1617 E. 4th, Santa Ana, Calif. **OALR**
Dlugasch, Louis, '29....110 E. 36th, New York 16, N. Y. **ALR**
Dobbs, O. R., '30, Comdr. (M.C.),
9927 Riverview Drive, St. Louis 15, Mo. **ALR**
①**Dodd, Oscar,** '03.....636 Church, Evanston, Ill. **OP**
Dodge, W. M., Jr., '34, Major (M.C.),
Glennan General Hospital, Okmulgee, Okla. **OP**
Doherty, Wm. B., '27....150 W. 55th, New York 19, N. Y. **OP**
Dolowitz, David A., '43, Lt. (M.C.), c/o A. Dolowitz,
5129 No. 12th st., Philadelphia, Pa. **ALR**
Donahue, Hugh C., '39
520 Commonwealth av., Boston 15, Mass. **OP**
Donlon, John J., '43, 112 Clinton st., Brooklyn, N. Y., also
21 S. Village av., Rockville Center, N. Y. **ALR**
Donnell, Herbert, '38....507 W. Main, Waxahachie, Texas **ALR**
Donnelly, Edward J., '38..1321 Spruce, Philadelphia 7, Pa. **OP**
②**Donoher, William D.,** '17
635 S. Westlake av., Los Angeles 5, Calif. **OALR**
Donohoe, Edward C., '31, Lt. Col. (M.C.), Sta. Hosp.,
A.A.F.P.S. (B), Minter Field, Bakersfield, Calif. **ALR**
Donovan, John E., '38.....265 W. 10th, Erie, Pa. **ALR**
Donovan, Richard Joseph, '43,
1400 Hancock, Quincy 69, Mass. **OALR**
Doran, Frank, '40.....Metz bldg., Grand Rapids 2, Mich. **ALR**
Dorge, Richard I., '22,
1111 Nicollet av., Minneapolis 2, Minn. **OALR**
Dougherty, Clifford L., '31.55 E. Washington, Chicago 2, Ill. **ALR**
Douglas, Edward M., '36
87-85—153rd, Jamaica 2, L. I., N. Y. **OP**
Douglas, Frederick A., '22
State Bank bldg., La Crosse, Wis. **OALR**
①**Dow, Frank E.,** '14.....4 West st., Northampton, Mass. **OP**
Dow, Julian Neal, '31,
9730 Wilshire blvd., Beverly Hills, Calif. **OP**
Dowling, Harvey E., '32, F/L R.C.A.F.,
414 4th av. W., Calgary, Alberta, Canada **OP**
①**Dowling, J. Ivimey,** '14
116 Washington av., Albany 6, N. Y. **OALR**
②**Dowling, J. Thomas,** '17.1115 Terry av., Seattle 1, Wash. **OALR**
Dowling, James Reginald, '40, Major (M.C.),
McCloskey General Hospital, Temple, Tex. **ALR**
Downey, Harold R., '38, Lt. Comdr. (M.C.),
1740 M st., N.W., Washington 6, D. C. **OP**
Downing, Arthur H., '43, Capt. (M.C.),
Medical Ind. Station, Fort Snelling 11, Minn. **OP**
Downing, James A., '36
Bankers Trust bldg., Des Moines 9, Iowa **ALR**
Drews, Leslie C., '43
516 Metropolitan bldg., St. Louis 3, Mo. **OP**
Driscoll, F. J., '33.....Reibold bldg., Dayton 2, Ohio **ALR**
Droegemueller, William H., '39, Major (M.C.),
116 Burnham Place, Evanston, Ill. **OP**
Drooker, J. C., '40, Major (M.C.), Ream Gen. Hosp.,
Box 9, Palm Beach, Fla. **ALR**
Druss, Joseph G., '36..1040 Park av., New York 28, N. Y. **ALR**
Dubins, Henry B., '43.....248 State, Albany 6, N. Y. **OP**
Dublin, George Joseph, '41
1930 Chestnut, Philadelphia 3, Pa. **OP**
Duboe, Sidney Harvey, '42, Capt. (M.C.), A.P.O. 923,
c/o Postmaster, San Francisco, Calif. **ALR**
Duehr, Peter A., '39
224 W. Washington av., Madison 3, Wis. **OP**
Duke, Roncie R., '31.....706 Franklin, Tampa 2, Fla. **ALR**
②**Dulaney, Octavus,** '17.....Brown bldg., Louisville 2, Ky. **ALR**
Dumas, Edward DeVan, '36
Medical Arts bldg., San Antonio 5, Texas **ALR**
Dunaway, Carl E., '35...Huntington bldg., Miami 32, Fla. **ALR**
Dunbar, John Charles, '40.Empire bldg., Pittsburgh 22, Pa. **OP**
Duncan, Perry E., '37.....101½ E. Main, Taylorville, Ill. **OP**
Duncan, R. A., '29.....Amarillo bldg., Amarillo, Texas **ALR**
③**Dundas-Grant, Sir James** (Retired).....England
Dunlap, Henry A., '37
7815 E. Jefferson av., Detroit 14, Mich. **OP**
Dunlap, Lawrence G., '20.....101 Main, Anaconda, Mont. **OALR**
Dunn, Austin G., '37
111½ Strongs av., Stevens Point, Wis. **ALR**
Dunnavan, Floyd Leighton, '42
Arts bldg., Vancouver, Wash. **OP**
Dunnington, J. H., '24.30 W. 59th, New York 19, N. Y. **OP-PI S**
Dunphy, Edwin B., '31, Lt. Comdr. (M.C.),
243 Charles st., Boston 14, Mass. **OP**
①**Duntley, G. S.,** '13.....Box 47, Macomb, Ill. **OALR**
Durr, S. A., '27..Medico-Dental bldg., San Diego 1, Calif. **OP**
①**Dutrow, H. V.,** '14, Lt. Col. (M.C.),
Lawson Gen. Hosp.,Atlanta, Ga. **OALR**
Dvorak, Joseph E., '37.Davidson bldg., Sioux City 13, Iowa **OP**
Dwyer, James G., '36...375 Park av., New York 22, N. Y. **ALR**
Dyar, Edward William, '40.Lt. (M.C.), Columbia City, Ind. **OP**
Dyer, B. G., '29.....109½ S. 4th, Manhattan, Kan. **ALR**
Dyer, C. P., '20.....Humboldt bldg., St. Louis 3, Mo. **OP**
and Garelock bldg., Webster Groves, Mo.
Dykman, Augustus B., '37
833 S.W. 11th av., Portland 5, Ore. **OP**
Dysart, Ben Robnett, '41
65 N. Madison av., Pasadena 1, Calif. **ALR**

E

Eagle, Watt W., '37.....Duke Hospital, Durham, N. C. **ALR**
Eagleton, W. P., '30.....15 Lombardy, Newark 2, N. J. **ALR**
Earl, Harry D., '36.....479 Sixth st., San Pedro, Calif. **ALR**
Earnest, Clarence E., '31.....Thatcher bldg., Pueblo, Colo. **ALR**
①**Easton, J. C.,** '04
First Nat'l Bank bldg., Springfield, Ohio **OALR**
Easton, Mahlon T., '39.....264 Beacon, Boston 16, Mass. **OP**
Ebeling, Wm. B., '30.....54 Greene av., Brooklyn 5, N. Y. **OP**
Eber, Carl Theo., '28, Carleton bldg., St. Louis 1, Mo. **OP**
Ebert, Reinhold O., '37.....64 Mt. Vernon, Oshkosh, Wis. **OP**
Edelman, Maurice I., '42, Lt. (M.C.),
849 Gunnison, Chicago, Ill. **ALR**
Edgerton, Ambrose E., '31
450 Sutter, San Francisco 8, Calif. **OP**
Edmunds, Stanley L., '38, Lt. (M.C.), U.S.N.R.,
13-D Hillside Road, Greenbelt, Md. **ALR**
①**Edwards, C. J.,** '13
First Nat'l Bank bldg., Vicksburg, Miss. **OALR**
Edwards, Gwilym Austin, '42
Home Guards bldg., Van Wert, Ohio **ALR**
Edwards, S. A., '35...Home Guards bldg., Van Wert, Ohio **ALR**
②**Edwards, Slocomb R.,** '18.107 S. Fifth, El Centro, Calif. **OALR**
Edwards, Thomas J., '38.330 Lowry bldg., St. Paul 2, Minn. **OP**
Egan, Burton W., '28.....408 North st., Logansport, Ind. **OALR**
Ehrlich, Max, '40, Capt. (M.C.),
721 N. Broad, Elizabeth 3, N. J. **ALR**
Ehrmann, Evelina W., '42
327 S. La Grange Road, La Grange, Ill. **OP**
Eimer, C. E., '28, Major (M.C.),
Medical & Psychol. Unit, Buckley Field, Colo. **ALR**
Elfman, Louis Kenneth, '42
6330 Lebanon av., Philadelphia 31, Pa. **ALR**
Ellenberger, Carl, '40.14805 Detroit av., Lakewood 7, Ohio **OP**
②**Elles, Norma B.,** '19
Niels Esperson bldg., Houston 2, Texas **OALR**
③①**Ellett, E. C.,** '98...Exchange bldg., Memphis 3, Tenn. **OP**
Elliff, Edgar Alonzo, '41.....216 N. 3rd, Sterling, Colo. **ALR**
③**Elliot, R. H.,** '13, Lt. Col., 54 Welbeck st.,
Cavendish sq. W., London, Eng.
Elliott, Alfred J., '43, S/L. C6610, R.C.A.F., overseas **OP**
Elliott, J. Norman, '38..Griesheim bldg., Bloomington, Ill. **ALR**
Elliott, Raymond E., '41
76 S. Fitzhugh, Rochester 8, N. Y. **ALR**
Ellis, Bert E., '31
303 Hume-Mansur bldg., Indianapolis 4, Ind. **ALR**
Ellis, C. Ward, '28.....215 N. Walnut, Lansing 15, Mich. **ALR**
Ellis, Orwyn H., '40, Lt. (j.g.), (M.C.),
647 Warner av., Los Angeles 24, Calif. **OP**
Elz, Jule T., '37....3606 Gravois blvd., St. Louis 16, Mo. **OP**
①**Emerson, Linn,** '06.....303 Park av., Orange, N. J. **OP**
Emery, John R., '38...Medical Centre, White Plains, N. Y. **ALR**
Emswiler, Herbert D., '37, 370 E. Town, Columbus 15, Ohio **ALR**
England, Welch, '36....1009 Market, Parkersburg, W. Va. **ALR**
Engler, Clarence Wm., '33
2323 Prospect av., Cleveland 15, Ohio **ALR**
Enlows, Ella M. A., '34
1816 R st. N.W., Washington 9, D. C. **ALR**
Epstein, Sidney S., '41, Capt. (M.C.),
Baxter Gen. Hosp., Spokane, Wash. **OP**
Equen, Murdock S., '31
144 Ponce de Leon av., N.E., Atlanta, Ga. **ALR**
Ernsting, Harry Carl, '42..8 W. Main st., Springfield, Ohio **OP**
Ersner, Matthew S., '20..1915 Spruce, Philadelphia 3, Pa. **ALR**
Eschenbrenner, John William, '37, Maj. (M.C.),
A.A.F., Station Hospital, Woodward, Okla. **ALR**
Esterman, Benjamin, '37.983 Park av., New York 28, N. Y. **OP**
Eubank, Ambrose E., '28.Bryant bldg., Kansas City 6, Mo. **OALR**
Eubank-Jones, Miriam D., '39, 944 Maple av., Evanston, Ill. **OP**
Evans, J. N., '25.....23 Schermerhorn, Brooklyn 2, N. Y. **OP**
Evans, John Doritte, '41, Major (M.C.),
Station Hospital, Craig Field, Selma, Ala. **ALR**
Evans, Maurice G., '32, 416 Marlborough, Boston 15, Mass. **ALR**
Evans, S. S., '32
Physicians & Surgeons bldg., Memphis 3, Tenn. **ALR**
Evans, Wm. H., '34, Lt. Comdr. (M.C.), U.S.N.R.,
c/o Fleet P. O., San Francisco, Calif. **ALR**
Evatt, Clay W., '40..112 Rutledge av., Charleston 16, S. C. **ALR**
Everett, A. Reginald, '39, 11 Wall st., New York 16, N. Y. **ALR**
Evers, Lorance Bixler, '41
University Hospital, Iowa City, Iowa **ALR**
①**Ewing, Fayette C.,** '99, (Retired)
"Nosegay," Pineville, La.

F

Fabricant, Noah D., '36.185 N. Wabash av., Chicago 1, Ill. **ALR**
Fackler, C. L., '30, Lt. Comdr. (M.C.),
U. S. Naval Reserve School, Northampton, Mass. **OALR**
Faggart, George H., '43.18 W. Oglethorpe av., Savannah, Ga. **ALR**
Faier, Herman I., '43, Major (M.C.),
Hoff General Hospital, Santa Barbara, Calif. **OP**
Faier, Samuel Z., '35, Capt. (M.C.), 0-283112
APO 515, c/o P.M., New York, N. Y. **OALR**
①**Fairbairn, John F.,** '12.925 Delaware av., Buffalo 9, N. Y. **ALR**
Fairchild, Nora M., '22
939 S. Figueroa st., Los Angeles 15, Calif. **OP**
Fairfax, Kenneth T., '39, Major (M.C.),
Station Hospital, Camp Croft, S. C. **OP**
Fairing, John W., '24.....122 N. Main, Greensburg, Pa. **OALR**
Falkson, Kurt F., '42.229 W. 78th st., New York 24, N. Y. **ALR**
②**Fallows, Howard D.,** '16
Foresters bldg., Mason City, Iowa **OALR**
Falls, Harold Francis, '43...Uni. Hosp., Ann Arbor, Mich. **OP**
②**Farmer, A. G.,** '19
Fidelity Medical bldg., Dayton 2, Ohio **OALR**
②**Farnsworth, M. A.,** '17
Opera House Apt. bldg., La Junta, Colo. **OALR**
Farrell, James I., '35
Lt. Comdr. (M.C.), 250 Genessee, Utica 2, N. Y. **OP**
①**Farrington, P. M.,** '04
89 Porter bldg., Memphis 3, Tenn. **ALR**
Farrior, J. Brown, '42, Capt. (M.C.),
A.P.O. 508, New York, N. Y. **ALR**
Fast, Ralph B., '36..136 E. Michigan, Kalamazoo 4, Mich. **ALR**
①**Fawcett, Ivan,** '13....75 Twelfth st., Wheeling, W. Va. **OALR**
Fein, Harry, '40.....483 Beacon, Boston 15, Mass. **ALR**
Feldman, Irvin, '39, Lt. Comdr. (M.C.), U.S.N.R.,
Post Med. Detachment, Marine Barracks, Parris Is., S. C. **ALR**
Feldman, Joseph, '40
668 Eastern Parkway, Brooklyn 13, N. Y. **ALR**
Feldman, Louis A., '35
259 New York av., Brooklyn 16, N. Y. **OP**
Fellows, M. Fording, '35, Comdr. (M.C.), U.S.N.R.,
Lion No. 6, San Bruno, Calif. **OP**
①**Fenton, R. A.,** '21, 1020 S. W. Taylor, Portland 5, Ore. **OALR**
Ferguson, E. R., '35.....404 Inman st., Cleveland, Tenn. **ALR**
Ferguson, George Burton, '40
1114 West Main st., Durham, N. C. **ALR**
Ferguson, R. J., '27.....59 College, New Haven 10, Conn. **OALR**
Fernandez, Luis J., '40, 15 Ponce de Leon av., Box 2206,
San Juan 10, Puerto Rico **OP**

Fernandez, Ricardo F., '39, 15 Ponce de Leon av., Box 2206, San Juan 10, Puerto Rico **OP**

Fernlund, G. B., '28 8620—115th, Richmond Hill, New York 18, N. Y. **ALR**

Ferris, Ralph G., '34.55 W. Maple av., Birmingham, Mich. **ALR**

Fewell, A. G., '31.....1924 Pine, Philadelphia 3, Pa. **OP**

Field, Homer Baker, '42, Capt. (M.C.), A.P.O. 923, c/o P. M., San Francisco, Calif. **OP**

Figi, Frederick A., '27....Mayo Clinic, Rochester, Minn. **LR-PI S**

Filmer, George Arnold, '41, Maj. (M.C.), William Beaumont General Hospital, El Paso, Tex. **OP**

Findlay, E. K., '30.....30 N. Michigan av., Chicago 2, Ill. **OP**

Fine, Abraham, '32...925 Ocean av., Brooklyn 26, N. Y. **OALR**

Fineberg, Maxwell, '30 607 N. Grand blvd., St. Louis 3, Mo. **ALR**

Fineberg, Nathan L., '38 475 Commonwealth av., Boston 15, Mass. **ALR**

Finegan, James F., '41, Lt. (M.C.), U.S.N.R., 6241 Underwood, Omaha 3, Nebr. **OP**

Fink, Leo W., '39.Medical Arts bldg., Minneapolis 2, Minn. **ALR**

Fink, Otto E., '38.....9 W. Madison, Danville, Ill. **OP**

Fink, Walter H., '27 Medical Arts bldg., Minneapolis 2, Minn. **OP**

Finney, Nancy E., '37..2525 Gilbert av., Cincinnati 6, Ohio **OP**

Firestone, Charles, '40 225 Meachann av., Elmont, L. I., N. Y. **ALR**

Fischbach, V. W., '31 Union Central bldg., Cincinnati 2, Ohio **ALR**

Fischer, Abraham, '39.Mercantile bldg., Rochester 4, N. Y **OP**

Fischer, Aubrey, '34 1835 Eye st. N.W., Washington 6, D. C. **ALR**

Fischer, N. Arthur, '25 Union Trust bldg., Pittsburgh 19, Pa. **ALR**

②**Fischer, V. B.**, '17.Mercantile Bank bldg., Boulder, Colo. **OALR**

①**Fisher, Carl** '12.1930 Wilshire blvd., Los Angeles 5, Calif. **OP**

Fisher, George Garthwaite, '41 271 New York av., Brooklyn 16, N. Y. **ALR**

Fisher, Gilbert Eugene, '42 Medical Arts bldg., Birmingham 5, Ala. **ALR**

Fisher, Guy Rothwell, '42 1 E. Beverley st., Staunton, Va. **ALR**

Fisher, H. Noland, '37....106 N. Silver st., Olney, Ill. **OP**

Fisher, James A., '25...501 Grand av., Asbury Park, N. J. **OALR**

Fisher, Lawrence Christian, '41..469 W. Market, York, Pa. **OP**

①**Fisher, W. A.**, '96.....156 N. Franklin, Chicago 6, Ill. **OALR**

Fishman, Louis Z., '35, Green's Eye Hospital, 1801 Bush, San Francisco 9, Calif. **ALR**

Fishof, Frank E., '41, Capt. (M.C.), Station Hospital, Camp Claiborne, La. **ALR**

Fitzgerald, Fred J. C., '34.118 E. 65th, New York 21, N. Y. **OP**

Fitzgerald, J. P., '25...30 N. Michigan av., Chicago 2, Ill. **OP**

Fitzgerald, J. R., '43...30 N. Michigan av.,Chicago 2, Ill. **OP**

Fitzgerald, William W., '37..27 Ludlow, Younkers, N. Y. also, 425 Madison av., New York, N. Y. **OP**

Fitz-Hugh, Glassell S., '39, Capt. (M.C.), A.P.O. 516, c/o P.M., New York, N.Y. **ALR**

Fitzhugh, William McP., Jr., '38, Lt. Comdr. (M.C.), 384 Post, San Francisco 8, Calif. **ALR**

Flack, J. Vincent, '37, Lt. Comdr. (M.C.), 90 Church, New York 7, N. Y. **OP**

Flaherty, Norman W., '39, Capt. (M.C.), Camp Dodge, Ia. **OP**

Flanders, Merton N., '42, Capt. (M.C.), Tilton General Hospital, Fort Dix, N. J. **ALR**

①**Flatley, T. J.**, '14.....1518 Fifth av., Moline, Ill. **OALR**

Flynn, Charles T., '27..41 Trumbull, New Haven 10, Conn. **ALR**

Flynn, James A., '24 1511 Rhode Island av. N.W., Washington 5, D. C. **ALR**

Flynn, J. Donald, '41, Lt. (M.C.), Naval Air Station (Dispensary), Pasco, Wash. **ALR**

Fogarty, Charles W., '20..372 St. Peter, St. Paul 2, Minn. **OALR**

Folken, Frank G., '28..201 S. Broadway, Albert Lea, Minn. **OALR**

Follweiler, Frank L., '43....423 York rd., Jenkintown, Pa. **ALR**

Folsom, W. H., '27.....11 N. Main, Fond du Lac, Wis. **OALR**

Font, Juan H., '33, P.O. Box 5363, Puerta de Tierra Station, Medical Arts bldg., San Juan, Puerto Rico **ALR**

Foote, William D., '43, Capt. (M.C.), Station Hospital, Hunter Field, Savannah, Ga. **OP**

Forbes, Sherman B., '22.....706 Franklin, Tampa 2, Fla. **OALR**

Ford, Hanby L., '28.....Robeson bldg., Champaign, Ill. **OALR**

Ford, Leroy Sargent, '42, Lt. (M.C.), 30 School st., Keene, N. H. **ALR**

Ford, William W., '38....109 E. Walnut, Green Bay, Wis. **ALR**

Forrester, Alex M., '43, Capt. (M.C.), Roswell Army Air Field, Station Hospital, Roswell, N. M. **ALR**

②**Forsythe, S. T.**, '19.....Rose bldg., Cleveland 15, Ohio **OALR**

Foster, Clarence Batchelder, '42, Maj. (M.C.), APO 376, c/o Postmaster, New York, N. Y. **OP**

Foster, George C., '36.....Fargo Clinic, Fargo, No. D. **OALR**

③①**Foster, Hal**, '94, (Retired), Hyde Park Hotel, Kansas City 2, Mo. **ALR**

Foster, Harold, '35 92-11 35th av., Jackson Hts., New York, N. Y. **ALR**

①**Foster, J. H.**, '13....1304 Walker av., Houston 2, Texas **ALR**

Foster, Warren H., '35....217 Fifth av. S., Clinton, Iowa **ALR**

Foster, Wayne J., '35....Higley bldg., Cedar Rapids, Iowa **ALR**

Fowler, Earle B., '25....55 E. Washington, Chicago 2, Ill. **OP**

Fowler, Edmund Prince, Jr., '40, Major (M.C.), 0-400230 APO 647, c/o PM., New York, N. Y. **ALR**

Fowler, Floyd J., '43, Akron Clinic, 513 W. Market, Akron 3, Ohio **ALR**

Fowler, James G., '37...412 Linwood av., Buffalo 9, N. Y. **OP**

Fowler, Robert H., '29..610 Park av., New York 21, N. Y. **ALR**

Fowlkes, John W., '29....12 E. 63rd, New York 21, N. Y. **ALR**

Fox, C. Calvin, '37.....1806 Spruce, Philadelphia 3, Pa. **ALR**

Fox, C. H., '20, Schuyler Hotel, 1017 Locust, Kansas City, Mo. **OALR**

Fox, Meyer S., '40 606 W. Wisconsin av., Milwaukee 3, Wis. **ALR**

Fox, Noah, '26.....841 E. 63rd, Chicago 37, Ill. **ALR**

Fox, Samuel L., '43...1820 Eutaw Pl., Baltimore 17, Md. **ALR**

Fox, Sidney A., '37, Major (M.C.), Newton D. Baker General Hospital, Martinsburg, W. Va. **OP**

Frackelton, Ralph J., '37 15701 Detroit av., Lakewood 7, Ohio **ALR**

Fralick, F. Bruce, '33 First National bldg., Ann Arbor, Mich. **OP**

Francis, Charles C., '39 610 West End av., New York 24, N. Y. **ALR**

Francis, Norton Lewis, '41, Lt. (M.C.), U.S.N.R., Navy 131, c/o Fleet P. O., San Francisco, Calif. **ALR**

Frank, David I., '37, Capt. (M.C.), 0-487642, A.P.O. 3658, c/o Postmaster, New York, N. Y. **ALR**

Frank, Herman W., '43.....Medical bldg., Gadsden, Ala. **OP**

①**Frank, Ira**, '05.....104 S. Michigan av., Chicago 3, Ill. **ALR**

Fraser, R. H., '25 25 W. Michigan av., Battle Creek, Mich. **ALR**

Fraser, Robert C., '29....940 Military, Port Huron, Mich. **ALR**

Fraser, W. G., '27.....Medical Arts bldg., Ottawa, Can. **OALR**

Frazee, John R., '36, Capt. (M.C.), 0-273819, A.P.O. 764, c/o Postmaster, New York, N. Y. **ALR**

Fread, Bernard, '37.....45 E. 85th, New York 28, N. Y. **OP**

Fred, Gustave B., '36....520 Beacon st., Boston 15, Mass. **ALR**

Freedman, A. O., '32 1396 St. Catherine st. W., Montreal, Can. **ALR**

Freedman, L. M., '20
475 Commonwealth av., Boston 15, Mass. **ALR**
Freeman, Sheldon B., '40
196 Linwood av., Buffalo 9, N. Y. **OP**
Freiman, George, '38..99 Lafayette av., Brooklyn 17, N. Y. **OP**
Freimuth, L. E., '27, Lt. Comdr. (M.C.),
U. S. Naval Hospital, Norman, Okla. **ALR**
①**French, R. F.**, '11..Masonic Temple, Marshalltown, Iowa **OALR**
French, Robert W., '37.....181 Pleasant, Malden, Mass. **OP**
Freund, E. M., '41.....762 Madison av., Albany 3, N. Y. **ALR**
Frey, Walter D., '43.....200 N. Upper, Lexington 15, Ky. **OP**
Frey, W. G., Jr., '34....121 E. 60th, New York 22, N. Y. **OP**
Friedberg, Stanton A., '39, Capt. (M.C.),
455 Barry av., Lincoln Park Sta., Chicago 14, Ill. **ALR**
①**Friedenwald, Harry**, '04
1212 Eutaw pl., Baltimore 17, Md. **OPA**
Friedenwald, J. S., '25..1212 Eutaw pl., Baltimore 17, Md. **OP**
Friedman, Benjamin, '33, Lt. Comdr. (M.C.),
201 W. 89th, New York, N. Y. **OP**
Friedman, Elmer Arthur, '41
6733 Newgard av., Chicago 26, Ill. **ALR**
Friedman, Louis Leonard, '40
6078 Jenkins Arcade, Pittsburgh 22, Pa. **ALR**
Friesner, Isidore, '36....101 E. 73rd, New York 21, N. Y. **A**
①**Fringer, W. R.**, '04.....109 Sheridan st., Rockford, Ill. **OP**
Fritsche, Theodore R., '37.Fritsche bldg., New Ulm, Minn. **OALR**
Frost, Albert D., '28.....150 E. Broad, Columbus 15, Ohio **OP**
Frost, Carl Grover, '41..333 Linwood av., Buffalo 9, N. Y. **ALR**
Frothingham, Edward, '28
8626 94th, Woodhaven 21, L. I., N. Y. **OALR**
Frothingham, Florie D., '41
133 E. 58th, New York 22, N. Y. **ALR**
Fry, W. E., '31.....1819 Chestnut, Philadelphia 3, Pa. **OP**
Fuchs, Valentine H., '28
American Bank bldg., New Orleans 12, La. **ALR**
Fuhring, Shirley Anthony, '41.....Address Unknown **ALR**
②**Fulkerson, C. B.**, '15
107 W. Michigan av., Kalamazoo 9, Mich. **OALR**
②**Fullenwider, C. M.**, '18...Barnes bldg., Muskogee, Okla. **OALR**
Fuller, John A., '28....First Nat'l Bank bldg., Reno, Nev. **OALR**
②**Fuller, T. E.**, '15.....209 Pine, Texarkana, Texas **OALR**
Fulton, Harry C., '37.....339 N. Duke, Lancaster, Pa. **OP**
Furlong, Thomas F., Jr., '35
Times Medical bldg., Ardmore, Pa. **ALR**
Furman, Martin A., '36, Major (M.C.),
Station Hospital, Camp Mackall, N. C. **ALR**
Furstenberg, A. C., '25
First Nat'l bldg., Ann Arbor, Mich. **ALR**
Fust, John H., '22.....231 W. 7th, Erie, Pa. **OALR**

G

Gabriel, C. K., '30..Illinois State Bank bldg., Quincy, Ill. **OALR**
Gadomski, Casimir Francis, '42
Lt. (M.C.), 331 S. Broad st., Elizabeth 2, N. J. **OP**
Gagion, Thomas Reed, '40.....23 Broad st., Pittston, Pa. **OP**
Gaida, Joseph B., '37....St. Mary's bldg., St. Cloud, Minn. **OP**
Gailey, Watson W., '22...1008 N. Main, Bloomington, Ill. **OALR**
Gaines, T. R., '40, Major (M.C.),
Finney General Hospital, Thomasville, Ga. **OP**
②**Gale, M. Jean**, '18.....Republic bldg., Denver 2, Colo. **OP**
Gallizzi, Vincent D., '42.....51 S. Main st., Pittston, Pa. **ALR**
Galloway, T. C., '27.....636 Church, Evanston, Ill. **ALR**
Gambill, Perry Judson, '42
New Jersey State Hospital, Greystone Park, N. J. **ALR**
Gamble, R. C., '29.....30 N. Michigan av., Chicago 2, Ill. **OP**
Gans, Jerome A., '43, Lt. (M.C.), A.U.S., 0-535443
Station Hospital, Fort Knox, Ky. **OP**
①**Garber, J. M.**, '11.....14 N. Mulberry, Mansfield, Ohio **OALR**
Garland, Frederick E., '34...483 Beacon, Boston 15, Mass. **LR**
Garlick, George B., '36.144 Golden Hill, Bridgeport 3, Conn. **ALR**
Garlock, Arthur Varney, '41....217½ 3rd, Bemidji, Minn. **OP**
Garnier, William Hampton, '41
Doctors bldg., Madisonville, Ky. **ALR**
Garofalo, Charles J., '37.439—77th st., Brooklyn 9, N. Y. **ALR**
Garrett, Ernest W., '35...Hanna bldg., Cleveland 15, Ohio **ALR**
Garrett, John D.....Bar Ass'n bldg., Indianapolis 4, Ind. **ALR**
Garrick, Thomas J., '36...40 E. 61st, New York 21, N. Y. **ALR**
Garten, Joseph Lambert, '40
Medical Arts bldg., Minneapolis 2, Minn. **ALR**
Gartner, Samuel, '39
1749 Grand Concourse, New York 53, N. Y. **OP**
Gaston, Ira E., '38
833 Medical-Dental bldg., Portland 5, Ore. **OALR**
Gatewood, E. T., '25.Professional bldg., Richmond 19, Va. **ALR**
②**Gatewood, W. L.**, '15
111 E. 61st st., New York 21, N. Y. **ALR**
Gaudet, Lucien S., '28.....405 Franklin, Natchez, Miss. **OALR**
Gaudreau, Honore E., '34..293 Bridge, Springfield 3, Mass. **OP**
Geary, Russell Diener, '42
337 Bridgeboro Road, Riverside, N. J. **ALR**
Geeseman, George Robert, '42, Capt. (M.C.),
Station Hospital, AAFTTC, Sioux Falls, So. Dak. **OP**
①**Geiger, C. W.**, '04.....Arcade bldg., Kankakee, Ill. **OALR**
Geiger, Dillon, '41, Major (M.C.),
N. Fee Lane, Bloomington, Ind. **ALR**
Gemeroy, Joseph C., '35
Henry Ford Hosp., Detroit 2, Mich. **OALR**
Gemmill, Walter D., '39.....130 Seventh, Monessen, Pa. **ALR**
George, Arthur R., '41, Capt. (M.C.),
Hdqrs. Second Air Force, Colorado Springs, Colo. **ALR**
George, Forney P., '37.....118 S. Hanover, Carlisle, Pa. **ALR**
Gernon, William, '41, Capt. (M.C.), 0-400253,
A.P.O. 924, c/o P. M., San Francisco, Calif. **OP**
Gerstley, Manfred J., '33, Comdr. (M.C.),
U. S. Naval Hospital, St. Albans 12, N. Y. **ALR**
Gerstner, Martin L., '40.468 Delaware av., Buffalo 2, N. Y. **ALR**
Gerzog, Bennett G., '37
891 St. Marks av., Brooklyn 13, N. Y. **ALR**
Gessler, Wm. F., '33
2902 Fairfield av., Ft. Wayne 6, Ind. **OALR**
Gewanter, Robert, '37
41-44 44th st., Long Island City 4, N. Y. **ALR**
Gibby, Harold J., '31.....27 Elm, Worcester 8, Mass. **ALR**
Gibson, Glen G., '35.....255 S. 17th, Philadelphia 3, Pa. **OP**
Gibson, William J., '43....650 Main, New Rochelle, N. Y. **ALR**
Gidoll, Sidney H., '42
516 Sutter st., San Francisco 2, Calif. **ALR**
Gifford, Harold, '37....Medical Arts bldg., Omaha 2, Nebr. **OP**
Gilbert, Joseph G., '36, Lt. Comdr. (M.C.),
Address Unknown **ALR**
Gilbert, Ralph H., '41..Metz bldg., Grand Rapids 2, Mich. **OP**
Gilfillan, Homer J., '41, Capt. (M.C.), 0-345048,
A.P.O. No. 700, c/o Postmaster, New York, N. Y. **ALR**
②**Gill, E. G.**, '19.....711 S. Jefferson, Roanoke 10, Va. **ALR**
Gill, Earl K., '36, Lt. Col. (M.C.), Station Hospital,
Majors Army Air Field, Greenville, Texas **ALR**
Gill, G. Bache, '23
1726 Eye st. N.W., Washington 6, D. C. **ALR**
Gill, Wm. Davis, '29
705 E. Houston, San Antonio 5, Texas **OALR**
Gillespie, Elmer H., '38....1101 Beacon, Brookline, Mass. **ALR**

Gillett, Wilbur G., '20...201½ N. Main, Wichita 2, Kan. **OP**
Gillette, D. F., '25.....State Tower, Syracuse 2, N. Y. **OP**
Gilligan, Alexander, '39....1154 Dean, Brooklyn 16, N. Y. **ALR**
Gilliland, O. S., '30.Professional bldg., Kansas City 6, Mo. **ALR**
Gillis, Marion Howard, Jr., '43, Maj. (M.C.),
405 Bush, Salisbury, Md. **OP**
Gillum, J. R., '20.....221 S. 6th, Terre Haute, Ind. **OALR**
Gilmore, Goodlatte Browne, '41
2940 Grand Concourse, New York 58, N. Y. **ALR**
Gilmore, Milton A., '36.....Box 172, Parkersburg, W. Va. **OP**
Gilmore, Robert O., '39.....1159 Hancock, Quincy, Mass. **ALR**
Gipner, J. F., '26...Medical Arts bldg., Rochester 7, N. Y. **OP**
Gissy, C. J., '43..717 Metropolitan bldg., St. Louis 3, Mo. **OP**
Gittins, T. R., '20.....Davidson bldg., Sioux City 13, Iowa **OALR**
Givner, Isadore, '36.....108 E. 66th, New York 21, N. Y. **OP**
Glasford, S. T., '24.....416 Elizabeth, Pekin, Ill. **OALR**
Glatt, Morris A., '31....55 E. Washington, Chicago 2, Ill. **ALR**
Gleason, John E., '40
David Whitney bldg., Detroit 26, Mich. **ALR**
Glick, Harry N., '28.3115 S. Grand blvd., St. Louis 18, Mo. **ALR**
①**Glosser, H. H.**, '12.....448 Franklin, Buffalo 2, N. Y. **OP**
Glover, Arthur C., '33.....311 W. Church, Elmira, N. Y. **OALR**
Glover, Lewis P., '38....1200 Fourteenth av., Altoona, Pa. **OP**
Glowacki, Ben F., '28...Maccabees bldg., Detroit 2, Mich. **OALR**
Glushak, Leopold, '38...1 West 85th, New York 19, N. Y. **ALR**
Goad, R. R., '31, Lt. Comdr. (M.C.),
4211 Farragut av., Hyattsville, Md. **OP**
Goar, Everett L., '26...1300 Walker av., Houston 2, Texas **OP**
Goehring, Donald Everett, '41
Butler S. & T. bldg., Butler, Pa. **ALR**
Goetzman, Arthur C., '42, Major (M.C.),
8907 Hennepin av., Niagara Falls, N. Y. **OP**
①**Goldbach, L. J.**, '11.....6 East Eager, Baltimore 2, Md. **OP**
Goldberg, Herman Krieger, '42
8 E. Eager st., Baltimore 2, Md. **OP**
Goldberg, Sol, '39, Major (M.C.),
La Garde Gen. Hosp., New Orleans 12, La. **OP**
Goldberg, Sylvan S., '35
1917 Wilshire blvd., Los Angeles 5, Calif. **ALR**
Goldcamp, Edward C., '32
Dollar Bank bldg., Youngstown 3, Ohio **ALR**
Goldcamp, John S., '41, Major (M.C.), 0-316784,
A.P.O. 922, c/o P. M., San Francisco, Calif. **OP**
Golden, Matthew G., '33.....1 Nevins, Brooklyn 17, N. Y. **ALR**
Goldenburg, Michael, '31
104 S. Michigan av., Chicago 3, Ill. **OP**
Golding, Joseph E., '25.....1 Nevins, Brooklyn 17, N. Y. **OP**
Goldmacher, H. B., '43.113 Elmora av., Elizabeth 2, N. J. **ALR**
Goldman, A. Milton, '28
1 N. Village av., Rockville Centre, L. I., N. Y. **OP**
Goldman, H. G., '23..57 W. 58th st., New York 19, N. Y. **ALR**
Goldman, Irving Brice, '40
121 E. 60th, New York, 22, N. Y. **ALR**
Goldsmith, Jacob, '41, Major (M.C.),
Station Hospital, Camp Blanding, Fla. **OP**
①②**Goldsmith, Perry**, '12
Medical Arts bldg., Toronto 5, Can. **ALR**
Goldstein, Henry Z., '36, Capt. (M.C.),
A.F.I.S. Camden Induction Center, Camden, N. J. **ALR**
Goldthwaite, Ralph H., '35, Brig. Gen. (M.C.),
Army and Navy Gen. Hosp., Hot Springs, Ark. **OALR**
Gollom, Joseph, '38.....425 Bloor st. W., Toronto, Can. **ALR**
Gomon, Louis D., '37.....Eddy bldg., Saginaw, Mich. **OP**
Gonne, Wm. S., '36
David Whitney bldg., Detroit 26, Mich. **ALR**
Gooch, John B., '35
Canal Bank bldg., New Orleans 12, La. **ALR**
Good, Palmer W., '37, Capt. (M.C.),
Hunter Field, Savannah, Ga. **OALR**
Good, Richard Peterson, '41, Major (M.C.),
Station Hospital, Fort Brady, Mich. **ALR**
Good, Wm. M., '29.....63 Center, Waterbury 18, Conn. **ALR**
Goodale, Robert L., '36..330 Dartmouth, Boston 16, Mass. **ALR**
Goodall, Edwin B., '34..71 Bay State rd., Boston 5, Mass. **OP**
Goode, Edward A., '29
143—28 Sanford av., Flushing, N. Y. **ALR**
Goodell, William, '34...121 Chestnut, Springfield 3, Mass. **ALR**
Goodfellow, Thomas J., '39
Arcade bldg., Saratoga Springs, N. Y. **OALR**
Goodhill, Victor, '42, 1101 Wilshire Medical bldg.,
1930 Wilshire blvd., Los Angeles 5, Calif. **ALR**
Goodman, E. Leonard, '36
1801 Eye st. N.W., Washington 6, D. C. **OP**
Goodman, Thomas L., '31
1601 Clover Lane, Fort Worth 7, Texas **ALR**
Goodspeed, Harry B., '34
36 Pleasant, Worcester 8, Mass. **ALR**
Goodwin, Thomas M., '38....Golden Clinic, Elkins, W. Va. **ALR**
Goodwin, Vernon L., '43
509 Central Tower, Youngstown 3, Ohio **ALR**
Goodyear, H. M., '24....Doctors bldg., Cincinnati 2, Ohio **ALR**
Gordon, Charles H., '26....46 Deering, Portland 4, Maine **ALR**
Gordon, Glenn G., '29.....Alexander bldg., Baker, Ore. **ALR**
②**Gordon, J. S.**, '19.....Bankers bldg., Milwaukee 2, Wis. **OALR**
Gordon, William, '33.....1721 Pine, Philadelphia 3, Pa. **ALR**
Gorin, Meyer, '42.....35 Chestnut, Rochester 4, N. Y. **ALR**
Gorman, James R., '36...Allied Arts bldg., Lynchburg, Va. **ALR**
Gorney, Arthur J., '39, Capt. (M.C.),
230 Boylston st., Boston, Mass. **ALR**
①**Gorrall, T. J. H.**, '09
16th and Vincennes, Chicago Heights, Ill. **OALR**
Gorilla, Laurence Vincent, '42
3115 S. Grand blvd., St. Louis 18, Mo. **OP**
Gosney, C. W., '20
5217 Hollywood blvd., Los Angeles 27, Calif. **OP**
Goss, Harold L., '37, Lt. Col. (M.C.), 0201505
A.P.O. 364, c/o P. M., New York, N. Y. **OP**
Gottlieb, Bernard N., '39
387 Eastern Parkway, Brooklyn 16, N. Y. **ALR**
Gough, Roy H., '31....600 W. 10th, Fort Worth 2, Texas **OALR**
Gould, Nathaniel Jay, '37
940 Grand Concourse, New York 56, N. Y. **OP**
Gouterman, Joseph I., '30, Major (M.C.), Sta. Hosp.,
Aberdeen Proving Ground, Md. **OP**
Goux, Raymond S., '22
David Whitney bldg., Detroit 26, Mich. **OALR**
Gowen, Thomas F., '39.....Address Unknown **ALR**
Gowens, Henry L., Jr., '42
2217 Delancey, Philadelphia 3, Pa. **OP**
Grace, Charles C., '33.....145 King, St. Augustine, Fla. **ALR**
Gracey, George F., '24.....209 State, Harrisburg, Pa. **OALR**
①**Gradle, Harry S.**, '11...58 E. Washington, Chicago 2, Ill. **OP**
Graebner, Herbert, '34...45 Park av., New York 16, N. Y. **ALR**
Graef, Fred W., '36....140 E. 54th, New York 22, N. Y. **ALR**
Granberry, Carl, '34
Maison Blanche bldg., New Orleans 16, La. **ALR**
Granger, W. B., '20.....Gazette bldg., Emporia, Kan. **OALR**
①**Grant, Fred E.**, '12
Kalamazoo Nat'l Bank bldg., Kalamazoo 9, Mich. **OALR**
Grant, H. W., '30.Lowry Med. Arts bldg., St. Paul 2, Minn. **OP**
Grant, Maurice David, '41
1860 Morris av., New York 53, N. Y. **ALR**
③**Graves, Basil**, '26
11 Devonshire pl., Upper Wimpole, W.1, London, Eng.

Greear, James N., Jr., '29, Lt. Col. (M.C.),
Valley Forge Gen. Hosp., Phoenixville, Pa. OP
Green, Hyman S., '35, Lt. Comdr. (M.C.), U.S.N.R.
6200 N. Kenmore av., Chicago 40, Ill. OALR
①Green, John, '05
3720 Washington blvd., St. Louis 8, Mo. OP
Greenberg, Maynard M., '39, Major (M.C.),
Station Hospital, Camp Crowder, Mo. OP
Greene, James S., '36....61 Irving pl., New York 3, N. Y. ALR
Greene, Laurence W., '28..Republic bldg., Denver 2, Colo. ALR
Greene, M. L., '30, Major (M.C.),
Station Hosp., Camp Swift, Texas OP
Greenfield, Samuel D., '27
169 New York av., Brooklyn 16, N. Y. ALR
Greenfield, William J., '28, 50 Anderson, Hackensack, N. J.,
and 30 E. 40th, New York 16, N. Y. ALR
Greenlee, Max R., '38, Lt. Comdr. (M.C.),
Acorn 45 Maritime bldg., Port Hueneme, Calif. ALR
Greenwood, G. J., '30...55 E. Washington, Chicago 2, Ill. ALR
Greever, B. L., '20.....100 N. Main, Hutchinson, Kan. OALR
Gregg, John B., '20
Security Nat'l Bank bldg., Sioux Falls, S. D. OALR
Gresser, Edward B., '29...39 E. 75th, New York 21, N. Y. OP
Griesemer, Z. Lawrence, '43,
1145 E. Jersey, Elizabeth 4, N. J. OP
Griesman, Bruno L., '39.1100 Park av., New York 28, N.Y. ALR
Griffey, Edward W., '29
Medical Arts bldg., Houston 2, Texas OALR
Griffey, Walter P., '39
Marine Hosp. Stapleton, New York, N. Y. OP
Griffin, Edwin A., '34
18 Prospect Park West, Brooklyn 15, N. Y. ALR
Griffith, Charles M., '36, Comdr. (M.C.),
c/o Fleet P. O., San Francisco, Calif. ALR
Griffith, Harold M., '30
Johnstown Bank & Trust bldg., Johnstown, Pa. OP
Grigsby, Roll O., '40....100 E. Second st., Ashland, Wis. ALR
Grimaldi, Americo John, '40
100 West 59th, New York 19, N. Y. OP
Grimmett, Roger Skelton, '40
406 Akron Saving & Loan bldg., Akron 8, Ohio ALR
Gross, Julius E., '20...3401 Fifth av., Pittsburgh 13, Pa. ALR
Grossman, Herman P., '38..210 Angell, Providence 6, R. I. OP
Grove, Bruce Alexander, '42.....141 E. Market, York, Pa. OP
Grove, Russell C., '37....60 E. 58th, New York 22, N. Y. ALR
①Grove, W. E., '14
324 E. Wisconsin av., Milwaukee 2, Wis. OALR
Gruppe, Karl W., '38.....258 Genesee, Utica 2, N. Y. ALR
Gsell, George Franklin, '38, Capt. (M.C.),
Sta. Hosp., Box 18, Lowry Field, Colo. OP
Gsell, J. F., '20.....911 Beacon bldg., Wichita 2, Kan. OALR
Gubner, Julius, '37.....25 Lefferts av., Brooklyn 25, N. Y. ALR
②Guggenheim, L. K., '18
1033 Gayley av., West Los Angeles 24, Calif. ALR
Guibor, George P., '33..30 N. Michigan av., Chicago 2, Ill. OP
Gulick, John Duncan, '41.704 Union, Schenectady 5, N. Y. ALR
Gump, Millard E., '39, Major (M.C.), A.A.F.A.F.S.,
M.A.A.F. Station Hospital, Marfa, Texas OP
①Gundelach, C. A., '12
University Club bldg., St. Louis 3, Mo. ALR
Gundersen, Trygve, '35, Maj. (M.C.), 0-400411,
A.P.O. 764, c/o Postmaster, New York, N. Y. OP
Gundrum, Lawrence Kramer, '42
1930 Wilshire blvd., Los Angeles 5, Calif. ALR
Gunn, Milus L., '29.....107 E. Mound, Harlan, Ky. ALR
Gurtov, John J., '37
1235 Grand Concourse, New York 52, N. Y. ALR
Gustafson, Olga Frideberg, '38
185 N. Wabash av., Chicago 1, Ill. OP
Guttman, M. Reese, '29.30 N. Michigan av., Chicago 2, Ill. ALR
Guy, Loren P., '34.....40 E. 62nd, New York 21, N. Y. OP
②Guyton, B. S., '18....512 Van Buren av., Oxford, Miss. OALR
Guyton, Jack Smallwood, '41
Johns Hopkins Hospital, Baltimore 5, Md. OP
Guzek, Joseph T., '43, Capt. (M.C.), 0-485618,
A.P.O. 425, c/o P. M., New York, N. Y. OP

H

Haas, Louis Richard, '42....Smith Clinic, Pittsburg, Kan. OALR
①Haden, Henry C., '13.....1914 Travis, Houston 3, Texas OP
Haessler, F. Herbert, '29...Wells bldg., Milwaukee 2, Wis. OP
Hagaman, Van Dyke, '34
Lamar Life bldg., Jackson 2, Miss. ALR
Hagan, Cornelius E., Jr., '42, Capt. (M.C.),
A.A.B. Hospital, Fort George Wright, Wash. ALR
Hageman, George Ricker, '42
Longmont Hospital, Longmont, Colo. ALR
Hagemann, John A., '25.Highland bldg., Pittsburgh 6, Pa. ALR
Hagens, Elmer William, '41.30 N. Michigan, Chicago 2, Ill. ALR
Haggart, Harry H., '37,
No. 1 Handasyde Lane, Cincinnati 8, Ohio ALR
Hagood, Daniel S., '43
822 First Nat'l Bank bldg., Montgomery 4, Ala. OP
Hague, Elliott B., '39.....454 Franklin, Buffalo 2, N. Y. OP
Hahn, Wm. H., '30.....15 Lombardy, Newark 2, N. J. OP
Hahn, William N., '38
City Nat'l Bank bldg., Omaha 2, Nebr. OP
Haik, George M., '43, Major (M.C.),
A.P.O. 428, c/o P. M., New York, N. Y. OP
Haines, Henry L., '43
Johns Hopkins Hospital, Baltimore 5, Md. ALR
Haire, Paul G., '35.....6 Pleasant, Malden, Mass. OP
Hale, Arthur S., '28.....Eaton Tower, Detroit 26, Mich. OALR
Haley, Paul J. D., '37.....333 Winthrop, Medford, Mass. ALR
Haley, Wm. T., '33.....531 Beacon, Boston 15, Mass. ALR
Hall, Alice K., '24.....25 E. Washington, Chicago 2, Ill. ALR
Hall, Colby, '42, Capt. (M.C.), 73rd Evacuation Hosp.,
National Military Home, Los Angeles 25, Calif. ALR
Hall, Edwin P., '28.....76 Chestnut, Oneonta, N. Y. ALR
①Hall, G. C., '07.....Brown bldg., Louisville 2, Ky. OALR
Hall, Lawrence H., '39....1520 Oak, So. Pasadena, Calif. OALR
Hall, Raymond A., '39, Capt. (M.C.),
3521 Hopkins rd., Youngstown, Ohio ALR
Hall, Sobisca S., '34, Lt. Col. (M.C.),
51st Gen. Hosp., Ft. Bliss, El Paso, Tex. ALR
Hallberg, Olav Erik, '42.....Mayo Clinic, Rochester, Minn. ALR
Hallum, Alton V., '34.....478 Peachtree, Atlanta 3, Ga. OP
②Halsted, Fred S., '18
Metropolitan bldg., Denver 2, Colo. OALR
Halton, Edward P., '37
1456 Northampton, Holyoke, Mass. ALR
Hamilton, Howard Eugene, '42
10515 Carnegie av., Cleveland 6, Ohio ALR
Hamilton, Richard G., '40
Medical Arts bldg., Pittsburgh 13, Pa. ALR
Hamlin, Fred E., '26..Medical Arts bldg., Roanoke 11, Va. ALR
Hammond, Arthur, '35
David Whitney bldg., Detroit 26, Mich. ALR
Hampsey, Joseph W., '37....May bldg., Pittsburgh 22, Pa. ALR
Hamrick, DeWitt, '40.....601 Jackson, Corinth, Miss. ALR
Hanckel, Richard White, Jr., '42
96-A Bull, Charleston 16, S. C. ALR
Hancox, Cecil W., '39..Bahr Arcade bldg., Pottstown, Pa. ALR

Hand, Frank, '35.....450 Sutter, San Francisco 8, Calif. **ALR**
Hands, Sidney G., '26.....201 W. 2nd, Davenport, Iowa **ALR**
Haney, Wm. P., '20...Medical Arts bldg., Omaha 2, Nebr. **OALR**
Hankins, George Geddy, '41
Medical Arts bldg., Newport News, Va. **ALR**
Hanley, James S., '37....40 W. 55th, New York 19, N. Y. **ALR**
Hanley, Jeanne Roeling, '39
Maison Blanche bldg., New Orleans 16, La. **ALR**
Hannon, R. Emmet, '42, Capt. (M.C.),
Station Hospital, Mitchel Field, L. I., N. Y. **OP**
Hans, C. L., '21..Provident Bank bldg., Cincinnati 2, Ohio **OALR**
Hansel, French K., '26.634 N. Grand av., St. Louis 3, Mo. **ALR**
Hansen, Erling W., '20
Medical Arts bldg., Minneapolis 2, Minn. **OP**
Hanser, Samuel Albert, '43, Lt. Col. (M.C.), 0-355900
A.P.O. 464, c/o P. M., New York, N. Y. **OP**
Hanson,, Wm. L., '29, Spivey bldg., East St. Louis, Ill. **ALR**
Hantman, Irvin, '39, Capt. (M.C.), A.S.C. Spokane
Army Air Field, Spokane, Wash. **ALR**
Happ, Linley Conrad, '41...280 Beacon, Boston 16, Mass. **ALR**
Hara, H. James, '33
Hinsdale Sanitarium and Hospital, Hinsdale, Ill. **ALR**
Haralson, Charles H., '29.Medical Arts bldg., Tulsa 3, Okla. **OP**
②**Harbert, John P.,** '19, 135 N. Main, Bellefontaine, Ohio **OALR**
Hardesty, J. F., '22
Missouri Theatre bldg., St. Louis 3, Mo. **OP**
Hardie, G. C., '30....290 W. Michigan av., Jackson, Mich. **OALR**
Harding, Myron S., '38
308 Hume-Mansur bldg., Indianapolis 4, Ind. **OP**
Hardy, Guerdan, '38, Lt. Comdr. (M.C.),
960 G av., Coronado, Calif. **OALR**
Hardy, LeGrand H., '28...23 E. 79th, New York 21, N. Y. **OP**
①**Hardy, W. F.,** '14.....Humboldt bldg., St. Louis 3, Mo. **OP**
Hardy, Walter B., '36.2121 Highland, Birmingham 5, Ala. **ALR**
Hargitt, Charles A., '26.17 Schermerhorn, Brooklyn 2, N. Y. **OP**
Harkins, Herbert P., '42, Lt. (M.C.), USNR,
Fleet P. O., San Francisco, Calif. **ALR**
Harkins, William Blake, '41, Lt. (M.C.),
c/o Fleet P.O., San Francisco 6, Calif. **ALR**
Harlowe, Harold Douglas, '40, Capt. (M.C.),
Station Hospital, Eglin Field, Fla. **ALR**
①**Harkness, Gordon F.,** '11
Putnam bldg., Davenport, Iowa **OALR**
Harned, John W., '39, Major (M.C.), Sta. Hosp.,
Truax Field, Madison 7, Wis. **ALR**
Harner, C. E., '39, Comdr. (M.C.),
Navy No. 128, c/o Fleet P. O., San Francisco, Calif. **OALR**
Harnick, Israel, '39...1132 Delaware av., Buffalo 2, N. Y. **ALR**
Harrell, Voss, '26.....Hofman bldg., Detroit 1, Mich. **ALR**
Harrill, James A., '41
Bowman Gray School of Medicine, Winston Salem, N. C. **ALR**
Harrington, John Thomas, '40, Lt. Comdr. (M.C.),
Navy 135, F.P.O., San Francisco, Calif. **ALR**
Harrington, Silas Frederick, '42
Medical Arts bldg., Dallas 1, Texas **ALR**
①**Harris, C. M.,** '10.Johnstown Trust bldg., Johnstown, Pa. **OALR**
Harris, Carl B., '41
319 Hume Mansur bldg., Indianapolis 4, Ind. **OP**
Harris, Edward W., '30, Major (M.C.),
Station Hospital, March Field, Calif. **ALR**
①**Harris, H. B.,** '10.Fidelity Medical bldg., Dayton 2, Ohio **OALR**
Harris, Harold Earl, '42.Cleveland Clinic, Cleveland 6, Ohio **ALR**
Harris, Herbert H., '39, Maj. (M.C.),
Mellie Esperson bldg., Houston 2, Texas **ALR**
Harris, J. Harley, '31
Methodist Hosp Doctors bldg., Memphis 4, Tenn. **ALR**
Harris, Morris L., '40.....20 Plaza, Brooklyn 17, N. Y. **ALR**
Harris, Robin, '23.....964 N. State, Jackson 6, Miss. **ALR**
Harrison, Fred H., '28
Second Nat'l Bank bldg., Connellsville, Pa. **OALR**
Hartenbower, George E., '38.Unity bldg., Bloomington, Ill. **ALR**
Hartgraves, Hallie, '38.28 W. Adams av., Detroit 26, Mich. **OP**
Hartman, Eugene C., '33
610½ Market, Parkersburg, W. Va. **OALR**
Hartshorne, Isaac, '26..30 W. 59th, New York 19, N. Y. **OP**
①**Hartzell, Sol M.,** '11...260 Tod Lane, Youngstown, Ohio **OALR**
①**Harvey, N. Darrell,** '04.112 Waterman, Providence 6, R.I. **OALR**
②**Haskell, Alfred W.,** '18.....142 High, Portland 3, Maine **OP**
①**Haskin, W. H.,** '13...375 Park av., New York 22, N. Y. **ALR**
Hasty, F. E., '25.....1203 Asturia, Coral Gables, Fla. **ALR**
①**Hatch, C. B.,** '08.....3 W. Church, Newark, Ohio **OALR**
Hatcher, William F., '38
409 Medical Arts bldg., Roanoke 11, Va. **OP**
②**Hauer, A. M.,** '16.....327 E. State, Columbus 15, Ohio **ALR**
①**Haughey, Wilfrid H.,** '11.Post bldg., Battle Creek, Mich. **OALR**
Hauser, I. Jerome, '38, Major (M.C.),
O'Reilly General Hospital, Springfield, Mo. **ALR**
Hauser, Maurice J., '43, Major (M.C.),
Station Hospital, Fort Custer, Mich. **OP**
Havens, Fred Z., '36....Mayo Clinic, Rochester, Minn. **ALR-PI S**
Hawkins, Raymond Willard, '40
39 S. Goodman, Rochester 7, N. Y. **ALR**
Hawman, Erie G., '20.4051 N. Broad, Philadelphia 40, Pa. **OP**
Hawthorne, Allen T., '38
110 N. Braddock, Winchester, Va. **ALR**
Hayden, Jerome William, '42
4458 Madison st., Chicago 24, Ill. **ALR**
Hayes, Merrill B., '40, Major (M.C.),
Station Hospital, Ft. Belvoir, Va. **ALR**
Hayes, Robert T., '36....42 Coburg, St. John, N. B., Can. **ALR**
Hays, Charles E., '39....U. S. Bank bldg., Johnstown, Pa. **ALR**
Hazen, S. Frank, '39.....231 Chestnut, Meadville, Pa. **ALR**
①**Heard, Mary K.,** '10
R.F.D. 2. Box 75, St. Petersburg 6, Fla. (Retired) **OP**
Heare, Louis C., '39, Lt. Col. (M.C.),
Station Hospital, Camp Howze, Tex. **OP**
Hearn, Paul P., '43, Capt. (M.C.),
Station Hospital B.A.A.F., Fort Myers, Fla. **ALR**
Heath, Parker, '24..David Whitney bldg., Detroit 26, Mich. **OP**
Hebble, Howard Miller, '40, Lt. (M.C.),
U. S. Naval Hospital, Philadelphia 45, Pa. **ALR**
Hebert, Arthur W., '27.450 Sutter, San Francisco 8, Calif. **OALR**
Hedemark, Norman G., '43, Major (M.C.),
Station Hospital, A.A.F.B.S., Big Spring, Texas **ALR**
Hedlund, G. O., '36, Lt. Comdr. (M.C.),
Navy 133, Fleet P.O., San Francisco, Calif. **ALR**
②**Heeb, Harry J.,** '18
212 W. Wisconsin av., Milwaukee 3, Wis. **OALR**
①**Heed, C. R.,** '10.....1205 Spruce, Philadelphia 7, Pa. **OP**
Heetderks, D. R., '30
Medical Arts bldg., Grand Rapids 2, Mich. **ALR**
Heidelman, George, '39
Union Central bldg., Cincinnati 2, Ohio **OP**
Heinberg, Charles J., '31...Medical Center, Pensacola, Fla. **ALR**
Heine, Lyman H., '38.....635 N. Main, Fremont, Nebr. **ALR**
Heine, William Howard, '41
Odd Fellow bldg., Fremont, Nebr. **ALR**
①**Heitger, J. D.,** '05.....Heyburn bldg., Louisville 2, Ky. **OALR**
Held, Albert E., '39, Major (M.C.),
A.P.O. 425, c/o P.M., New York, N. Y. **ALR**
Hempstead, Bert E., '20....Mayo Clinic, Rochester, Minn. **ALR**
Henderson, L. E., '30.....129 Clinton, Watertown, N. Y. **OP**
Hendricks, Louis J., '31...Carew Tower, Cincinnati 2, Ohio **OP**

Henner, Robert, '37, Capt. (M.C.),
Barksdale Field, Shreveport, La. **ALR**
Hennessey, Wm. W., '20.....333 Essex, Salem, Mass. **OALR**
①**Henning, Carl,** '08
1752 Massachusetts av. N.W., Washington 6, D. C. **OP**
①**Henninger, L. L.,** '13
65 N. Madison av., Pasadena 1, Calif. **OALR**
Henry, George Arnold, '40, Major (C.A.M.C.),
Canadian Army Overseas **ALR**
Henry, L. L., '43.....595 E. Colorado, Pasadena 1, Calif. **OP**
Henry, Robert, '42, Capt. (M.C.),
322 E. James, Colfax, Wash. **OP**
Hensle, Otto S., '40, Major (M.C.),
100 Prospect av., Hackensack, N. J. **ALR**
②**Henton, George E.,** '18
812 S.W. Washington, Portland 5, Ore. **OP**
Henton, George H., '37, Lt. Col. (M.C.),
Williams Field, Chandler, Ariz. **OP**
Herr, Albert H., '37, Major (M.C.),
212 Steiner bldg., Lima, Ohio **ALR**
②**Herriman, L. L.,** '18...Physicians' bldg., Alamosa, Colo. **OALR**
Hert, Cecil Benedict, '40, Major (M.C.), Ashford
General Hosp., White Sulphur Springs, W. Va. **ALR**
Hertz, William J., '36.....125 N. 8th, Allentown, Pa. **ALR**
Herzig, Arthur J., '41.667 Madison av., New York 21, N. Y. **ALR**
Herzog, Max, '41, Capt. (M.C.),
Station Hosp., Fort Jackson, S. C. **OP**
①**Hess, Wm. L.,** '04
1610 Sawtelle blvd., West Los Angeles, Calif. **OALR**
Hetrick, J. A. Werner, '38
667 Madison av., New York 21, N. Y. **ALR**
Hetrick, L. E., '23.....40 E. 61st, New York 21, N. Y. **ALR**
and 260 North av., New Rochelle, N. Y.
①**Hetzel, C. C.,** '06.....Eccles bldg., Ogden, Utah **OALR**
Hewson, Wm., '31....2031 Locust st., Philadelphia, Pa. **ALR**
Hickey, Harold L., '30.....Republic bldg., Denver 2, Colo. **ALR**
Hickman, Warren R., '34, Capt. (M.C.),
Broadway Hotel, Cincinnati, Ohio **ALR**
Hicks, Vonnie M., '26....Professional bldg., Raleigh, N. C. **OP**
Hicks, William J., '30.....15 King, Middletown, N. Y. **ALR**
Hicks, William Wynne, '42
202 E. 6th st., Ellensburg, Wash. **ALR**
Higbee, D. R., '27....3245 Fourth av., San Diego 3, Calif. **ALR**
Higgins, R. P., '24.....20 Court, Cortland, N. Y. **OALR**
①**Higgins, S. G.,** '11....411 E. Mason, Milwaukee 2, Wis. **OALR**
High, Clifton E., '35....Combs-Worley bldg., Pampa, Texas **OP**
High, Isaac B., '31.....326 N. 5th, Reading, Pa. **ALR**
Hilding, Anderson C., '31
Medical Arts bldg., Duluth 2, Minn. **OALR**
Hildreth, H. Rommel, '34
Metropolitan bldg., St. Louis 3, Mo. **OP**
Hilgartner, Henry L., Jr., '35, Major (M.C.),
A.P.O. 511, c/o P. M., New York, N. Y. **OP**
②**Hill, C. E.,** '19.....160 Bloor st. W., Toronto 5, Can. **OP**
Hill, Emerson S., '37.....51 Main, Torrington, Conn. **ALR**
Hill, Frederick T., '28.Professional bldg., Waterville, Maine **ALR**
Hill, Howard E., '39, Major (M.C.), Sta. Hosp.,
A.A.F.T.S., 1660 E. Hyde Park blvd., Chicago 15, Ill. **ALR**
Hill, Howard F., '28.....177 Main, Waterville, Maine **OP**
Hilleman, George A., '40
267 Alexander st., Rochester 7, N. Y. **ALR**
Hillyer, Ernest C., '43, Comdr. (M.C.),
c/o Fleet P. O., San Francisco, Calif. **OP**
Himelfarb, H. M., '41, Capt. (M.C.),
R. D. No. 4, Chambersburg, Pa. **ALR**
Hinderer, Kenneth H., '39, Capt. (M.C.),
Station Hosp., Camp Forrest, Tenn. **ALR**
Hipskind, Myron Maxwell, '40
6060 Drexel av., Chicago 37, Ill. **ALR**
Hirschfelder, Max Kurt, '41....408 W. 2nd, Centralia, Ill. **OP**
Hirschmann, Hans A., '39
1502 Mott av., Far Rockaway, N. Y. **ALR**
Hirst, Otto C., '32.....255 S. 17th, Philadelphia 3, Pa. **ALR**
Hitschler, William J., '39
8840 Germantown av., Philadelphia 18, Pa. **ALR**
①**Hitz, H. B.,** '02.....411 E. Mason, Milwaukee 2, Wis. **ALR**
Hitz, John B., '34, Maj. (M.C.), A.A.F.N.S., Hondo, Tex. **OP**
Ho, Wayne Y. H., '37, Capt. (M.C.), 0-534488, Sta. Hosp.,
Camp Swift, Bastrop, Texas **ALR**
Hobart, Carl, '29.....Metropolitan bldg., St. Louis 3, Mo. **OP**
Hochfilzer, John J., '35
Lowry Med. Arts bldg., St. Paul 2, Minn. **ALR**
Hodge, George E., '30....1414 Drummond, Montreal, Can. **ALR**
Hodge, James V., '39.....105 Market, Kingsport, Tenn. **ALR**
②**Hodsdon, B. F.,** '18.....P.O. Box 923, Miami 6, Fla. **OALR**
Hoeltgen, Maurice M., '43...1607 W. 51st, Chicago 9, Ill. **OP**
Hoffman, L. G., '27....30 N. Michigan av., Chicago 2, Ill. **OALR**
Hoffman, N. Fulmer, Jr., '43
2035 Delancey pl., Philadelphia 3, Pa. **ALR**
Hoffman, Walter F., '20....1317 Marion, Seattle 4, Wash. **OP**
Hoffman, Walter Lees, '39
Medical Arts bldg., Minneapolis 2, Minn. **OP**
Hoffman, William A., '32
30 N. Michigan av., Section 2, Chicago 2, Ill. **OP**
Hoffman, John N., '27....405 Brant bldg., Canton 2, Ohio **ALR**
Hofling, Charles A., '29.....707 Race, Cincinnati 2, Ohio **OP**
Hofmann, Albert P., '36.....630 Vine, Cincinnati 2, Ohio **ALR**
Hofmann, W. P., '25.....Kahl bldg., Davenport, Iowa **OALR**
Hogan, Chester F., '41, Lt. Comdr. (M.C.),
Mayo Foundation, Rochester, Minn. **ALR**
Hogan, W. L., '24.....750 Main, Hartford 3, Conn. **OP**
Hogsett, Smith Fuller, Jr., '42, Lt. (M.C.), U.S.N.R.
3009 Erie st. S.E., Apt. B-263, Washington 20, D. C. **OP**
①**Hogshead, J. M.,** '12
602 Georgia av., Chattanooga 3, Tenn. **OALR**
①**Hogue, D. W.,** '08.....34 W. High, Springfield, Ohio **OALR**
Hogue, John Dan, '41....909 Lexington av., Altoona, Pa. **ALR**
②**Hohf, Julius A.,** '15.....Clinic bldg., Yankton, S. D. **OALR**
Holcombe, Virgil Eugene, '40
Medical Arts bldg., Charleston 1, W. Va. **ALR**
Holden, Harold M., '36
514 N. Linden Dr., Beverly Hills, Calif. **ALR**
Holden, William H., '39...3 E. 85th, New York 28, N. Y. **ALR**
Holding, Bruce F., '36.....Bell bldg., Montgomery 4, Ala. **ALR**
Holinger, Paul H., '37..700 N. Michigan, Chicago 11, Ill. **L**
Hollabaugh, Charles F., '38
Shackleford Rd., Nashville 4, Tenn. **OP**
Holland, Emory E., '33.....51 S. 8th, Richmond, Ind. **OP**
Holland, John Gill, '41.....725 Church, Lynchburg, Va. **ALR**
Hollender, Abraham R., '31
307 N. Michigan av., Chicago 1, Ill. **ALR**
Holloway, J. Minor, '32
1200 Prince Edward, Fredericksburg, Va. **ALR**
Hollowell, Frederick Dixon, '40
Lamar Life bldg., Jackson, Miss. **ALR**
Holmen, Robert W., '43
Lowry Medical Arts bldg., St. Paul 2, Minn. **ALR**
Holmes, Edgar M., '36..330 Dartmouth, Boston 16, Mass. **ALR**
Holmes, Will W., '35, Col. (M.C.),
Station Hospital, Camp Croft, S. C. **ALR**
Holohan, Joseph L., '37.....330 State, Albany 6, N. Y. **ALR**
Holstein, Theodore, '42, Capt. (M.C.), 0-1702652
A.P.O. 9826, c/o P. M., New York, N. Y. **OP**

①**Holt, E. Eugene, Jr.,** '12.723 Congress, Portland 4, Maine **OP**
Holt, Helen, '43.....720 N. Michigan av., Chicago 11, Ill. **OP**
Holzberger, John C., '32
7005 Freshpond, Brooklyn 27, N. Y. **ALR**
Holzer, Wm. F., '31.....36 Pleasant, Worcester 8, Mass. **OP**
Hombach, Frank Joseph, '38
1421 State, Santa Barbara, Calif. **OP**
②**Hompes, J. J.,** '15.....Sharp bldg., Lincoln 8, Nebr. **OALR**
Honiss, John R., '37....277 Alexander, Rochester 7, N. Y. **ALR**
Hooker, James F., '39, Capt. (M.C.), U.S.N.,
Navy 121, Box 45, c/o Fleet P. O., New York, N. Y. **ALR**
Hoople, Gordon D., '29, Major (M.C.),
Medical Arts bldg., Syracuse 2, N. Y. **ALR**
Hooss, C. H. A., '30
3115 S. Grand blvd., St. Louis 18, Mo. **OP**
Hooval, John Harold, '41....105 W. C st., Ontario, Calif. **ALR**
Hoover, Walter B., '34
605 Commonwealth, Boston 16, Mass. **ALR**
Hope, Robert M., '37.108 Rutledge av., Charleston 16, S.C. **ALR**
Hopkins, G. H., '30.....702 N. Main, Pueblo, Colo. **OP**
Hornback, George A., '35....500 Broadway, Hannibal, Mo. **ALR**
Hornbogen, Daniel Powell, '41, Lt. Comdr. (M.C.),
Great Lakes Naval Hospital, Great Lakes, Ill. **OP**
Horner, Warren D., '35, Capt. (M.C.),
2518 Jackson st., San Francisco, Calif. **OP**
Hory, Joseph S., '39
2488 Grand Concourse, New York 58, N. Y. **OP**
Hotchkiss, Walter T., '31
541 Lincoln rd., Miami Beach 39, Fla. **ALR**
Houlahan, James M., '36, 117 S. 2nd av., Mt. Vernon, N. Y.
and 653 Park av., New York, N. Y. **OP**
Hourn, George E., '20
University Club bldg., St. Louis 3, Mo. **ALR**
House, Howard Payne, '41
511 S. Bonnie Brae, Los Angeles 5, Calif. **ALR**
House, Hugh O., '38
1835 Eye st. N.W., Washington 6, D. C. **ALR**
House, Leland Richmond, '40
312 N. Boyle av., Los Angeles 33, Calif. **ALR**
Houser, Ben P., '42, Capt. (M.C.),
18 W. Holland, Summit Hill, Pa. **ALR**
Houser, Karl M., '34...2035 Delancey, Philadelphia 3, Pa. **ALR**
Howard, H. Sargent, '43.418 Jefferson bldg., Peoria 2, Ill. **ALR**
Howard, Harvey J., '28.Park Plaza Hotel, St. Louis 8, Mo. **OP**
①**Howard, J. W.,** '12, (Retired),
Residence 6026 McGee, Kansas City 2, Mo. **OALR**
Howard, John Clair, Jr., '42, Lt. (j. g.) (M.C.),
Professional bldg., Kansas City 6, Mo. **ALR**
Howard, Lloyd G., '23....Park bldg., Council Bluffs, Iowa **OALR**
Howard, Robert E., '31
Union Central bldg., Cincinnati 2, Ohio **ALR**
Howard, William H., '23, Capt. (M.C.),
3949 Portland av., Minneapolis 7, Minn. **OALR**
Howard, William H., '37
9152 Commercial av., Chicago 17, Ill. **OP**
Howard, William M., '38.389 Linwood av., Buffalo 9, N. Y. **OP**
Howarth, Ervin McIntyre, '40, c/o Mrs. M. Verhusen
4126 W. 61st, Los Angeles, Calif. **ALR**
Howe, A. W., '22....Medical Arts bldg., Tacoma 2, Wash. **OALR**
Howe, Spencer S., '29
Bellingham Nat'l Bank, Bellingham, Wash. **OALR**
Howell, Donald M., '31, Wiechmann bldg., Saginaw, Mich. **ALR**
Howell, Homer Preston, '42, Capt. (M.C.),
Station Hosp., Hamilton Field, Calif. **OP**
Howell, Stacy C., '39
478 Peachtree st. N.E., Atlanta 3, Ga. **OP**
Howland, Alvin Wilcox, '39, Major (M.C.),
3310 Fairmount av., Atlantic City, N. J. **OP**
Hoxie, Derrick, '43, Lt. Comdr. (M.C.), U. S. Naval
Receiving Barracks, Shoemaker, Calif. **ALR**
Hubbard, Wm. B., '31..302 Paterson bldg., Flint 3, Mich. **OALR**
Hubble, William Fields, '42.Citizens bldg., Decatur 30, Ill. **ALR**
Huber, Walter A., '32...Medical Arts bldg., Tulsa 3, Okla. **OP**
Hudock, John Paul, '41..67 Broad st., Johnson City, N. Y. **ALR**
Hughes, L. W., '25.....965 Broad, Newark 2, N. J. **OP**
Hughes, Ray W., '28
David Whitney bldg., Detroit 26, Mich. **OALR**
Hughes, T. E., '22.....1000 W. Grace, Richmond 20, Va. **ALR**
①**Hughes, W. F.,** '11
401 Hume-Mansur bldg., Indianapolis 4, Ind. **OP**
Hughes, Wendell L., '29
Professional bldg., Hempstead, N. Y. **OP-PI S**
Hughes, William F., Jr., '43
619 N. Washington, Baltimore 5, Md. **OP**
Hughes, William Lauch, '40..Lamar bldg., Jackson 2, Miss. **ALR**
Hughson, Walter, '37.Abington Mem. Hosp., Abington, Pa. **A**
Hulka, J. H., '34, 30-28 36 st., Long Island City 3, N. Y. **OALR**
Hull, Allen, '35.....401 76th, Brooklyn 9, N. Y. **OP**
Hunnicutt, Leland G., '38, Major (M.C.),
Station Hospital, Kearns, Utah **ALR**
Hunt, Guy A., '36..Butler Co. Nat'l Bk. bldg., Butler, Pa. **OP**
Hunt, Thomas Ewell, '39
First Nat'l Bank bldg., Paris, Texas **ALR**
Hunt, Westley M., '29..907 Fifth av., New York 21, N. Y. **ALR**
Hunt, William Remfry, '38, Lt. Comdr. (M.C.),
Section Base, San Diego 49, Calif. **ALR**
Hunt, Wm. T., Jr., '34.....514 Penn, Huntingdon, Pa. **OALR**
Hunter, J. F., '30.....503 Penn av., Turtle Creek, Pa. **ALR**
Hunter, R. J., '30, Lt. Comdr. (M.C.), U.S.N.R.,
U. S. Naval Base, Cape May, N. J. **ALR**
Hurd, Allan C., '40, Lt. (M.C.), U.S.N.R., Navy 221,
c/o Fleet P. O., NewYork, N. Y. **ALR**
Hurd, Fritz D., '39.Medical Arts bldg., Great Falls, Mont. **ALR**
①**Hurd, L. M.,** '10.....Box F, Rawayton, Conn. **ALR**
②**Hurff, J. Wallace,** '19.....671 Broad, Newark 2, N. J. **OALR**
Hurst, V. Reeves, '22.....315 N. Center, Longview, Texas **OALR**
Hurwitz, Paul, '43, Major (M.C.),
Station Hospital, Camp Ellis, Ill. **OP**
Husik, D. N., '21.....1820 Spruce, Philadelphia 3, Pa. **ALR**
Hutcherson, John Kenneth, '41
Francis bldg., Louisville 2, Ky. **OP**
Hutchinson, Abbott T., '28
4086 Roynolds av., Coconut Grove, Fla. **ALR**
Hutchinson, Ben B., '38, Capt. (M.C.),
Station Hospital, SAAAB, Santa Ana, Calif. **OP**
Hutchison, K. O., '30
1509 Sherbrooke st. W., Montreal, Can. **ALR**
Hutchison, Lloyd Snyder, '42, Lt. Comdr. (M.C.),
U.S.N.R., U. S. Naval Hosp., Paris Island, S. C. **ALR**
Hyman, Morris, '36, Capt. (M.C.), 1139 Towanda
Terrace, Bond Hill, Cincinnati 16, Ohio **ALR**
Hymes, Charles, '39
Medical Arts bldg., Minneapolis 2, Minn. **OP**
Hynes, Frank Joseph, '41.121 E. 60th, New York 22, N. Y. **ALR**

I

Ide, David, '39.1749 Grand Concourse, New York 53, N. Y. **ALR**
①**Iglauer, Samuel,** '04...707 Race, Cincinnati 2, Ohio **ALR-PI S**
①**Imperatori, C. J.,** '11.108 E. 38th, New York 16, N. Y. **ALR**
Ingalls, Raymond George, '41
73 East 71 st., New York 21, N. Y. **OP**

①Ingersoll, J. M., '08
1700 S. Bayshore Lane, Miami 33, Fla. ALR
①Inglis, Harry J., '14..43 Bay State rd., Boston 15, Mass. ALR
Ingram, Hollis Carlyle, '40, Capt. (M.C.),
842 Laurel, Orlando, Fla. OP
②Ingram, L. C., '15.....842 Laurel, Orlando, Fla. OALR
Insel, Herman Herbert, '42, Lt. (M.C.),
Navy No. 416, c/o Fleet Post Office, New York, N. Y. ALR
Ireland, Percy E., '36, Maj. (RCAMC),
Canadian Army Overseas ALR
Irgens, Edwin R., '38, Major (M.C.), Station Hospital,
Fort Williams, Maine ALR
②Irvine, A. Ray, '18, Room 700,
727 West 7th st., Los Angeles 14, Calif. OP
Irvine, Robert S., '29...490 Post, San Francisco 2, Calif. OALR
Irvine, S. R., '38, Capt. (M.C.),
Station Hospital, Hammer Field, Fresno, Calif. OP
Irwin, John Joseph, '42
Baronne bldg., New Orleans 12, La. ALR
Irwin, Thomas M., '42, Major (M.C.),
Thayer General Hospital, Nashville, Tenn. ALR
Irwin, Vincent J., Jr., '21..1634 Main, Springfield 3, Mass. OALR
Irwin, Willard James, '42, Capt. (M.C.), Address Unknown ALR
Isbell, D., '40.....552 McCallie av., Chattanooga 3, Tenn. ALR
Isbell, Euclid A., '43.....948 Forrest av., Gadsden, Ala. ALR
①Ivins, H. M., '10..R.F.D. 4, Box 36, Santa Cruz, Calif. OALR
Ivins, William Clifford, '40..455 W. State, Trenton 8, N. J. ALR
Ivy, Robert H., '34..1930 Chestnut, Philadelphia 3, Pa. ALR-PI S

J

Jackson, Atras E., '28
Medical Arts bldg., Ft. Worth 2, Texas OP
①Jackson, Chevalier, '04..255 S. 17th, Philadelphia 3, Pa. L
Jackson, Chevalier L., '34..255 S. 17th, Philadelphia 3, Pa. L
Jackson, Clarence C. R., '37
Lakeside Hospital, Cleveland 6, Ohio ALR
Jackson, J. D., '20.....232 W. 8th, Erie, Pa. OALR
Jackson, Rufus, '37
Louisiana Nat'l Bank bldg., Baton Rouge 6, La. ALR
Jacobius, Lawrence, '39
2021 Grand Concourse, New York 53, N. Y. ALR
Jacobs, Clyde Hillock, '41
Geisinger Memorial Hospital, Danville, Pa. OP
Jacobs, Milton, '33....122 S. Michigan av., Chicago 3, Ill. OP
①Jacobs, S. M., '13.....15 W. 81st, New York 24, N. Y. OALR
①Jacoby, Douglas P. A., '12...106 Touro, Newport, R. I. OAL
Jacoby, Mark W., '33.....Hanna bldg., Cleveland 15, Ohio OP
Jaffe, Benjamin, '39, Capt. (M.C.),
Harmon General Hospital, Longview, Texas OP
Jaffe, Morris, '42
940 Grand Concourse, New York 56, N. Y. OP
James, George R., '37
Wyoming Nat'l Bank bldg., Casper, Wyo. OP
James, Voyle, '40..6253 Hollywood blvd., Hollywood, Calif. ALR
James, Wm. M., '31...508 N. Grand av., St. Louis 3, Mo. OP
Jamison, Edgar L., '41, Capt. (M.C.), 0-3071192,
A.P.O. 928, c/o P.M., San Francisco, Calif. ALR
Janse, Hector M., '37.....3717 Main, Houston 4, Texas ALR
Jarrott, James T., '43...131 Fulton av., Hempstead, N. Y. OP
Jarvis, DeForest C., '29....Quarry Bank bldg., Barre, Vt. ALR
①Jean, George W., '22
2774 Puesta del Sol Road, Santa Barbara, Calif. OP
Jellis, Walter, '41, Lt. Comdr. (M.C.),
Princeton University, Princeton, N. J. ALR
Jenkins, Albert H., '39, Major (M.C.), 0-1821216,
A.P.O. 9788, c/o P.M., San Francisco, Calif. ALR
Jennings, Charles W., '31
121 University pl., Pittsburgh 13, Pa. OP
Jensen, August Frithiof, '40
322 De Mers av., Grand Forks, N. Dak. OP
Jensen, Carl D., '39, Lt. (M.C.),
Medical and Dental bldg., Seattle 1, Wash. OP
Jerome, Bourne, '43, Major (M.C.),
Station Hospital, Fort Sheridan, Ill. OP
Jesberg, Simon, '32
500 S. Lucas av., Los Angeles 13, Calif. ALR
Jessel, Erwin, '42, Capt. (M.C.),
204 West 14th, New York, N. Y. OP
①Jobson, G. B., '08.West Park and Liberty, Franklin, Pa. OALR
Johns, Juanita Pearl, '33
313 Commonwealth av., Boston 15, Mass. OP
Johnson, Charles I., '39, Comdr. (M.C.), Navy 156,
c/o Fleet P. O., San Francisco, Calif. ALR
Johnson, David M., '32, Lt. Comdr. (M.C.), Naval College
Training Unit, Uni. of Wash., Seattle 5, Wash. ALR
Johnson, Don, '39.1835 Eye st. N.W., Washington 6, D. C. ALR
Johnson, Ernest D., '38....317 W. Main, Alhambra, Calif. OALR
Johnson, Gayle T., '39, Major (M.C.),
Station Hospital, Robins Field, Ga. ALR
①Johnson, H. R., '13.....320 Jefferson, Fairmont, W. Va. OALR
Johnson, Henrietta M., '32
595 E. Colorado, Pasadena 1, Calif. OP
Johnson, Henry P., '35.....32 Deering, Portland 3, Maine ALR
Johnson, Leighton F., '37
29 Bay State road, Boston 15, Mass. ALR
Johnson, Lorand V., '38
Lakeside Hospital, Cleveland 6, Ohio OP
Johnson, Malcolm R., '41, Lt. Col. (M.C.),
Station Hospital, Camp Crowder, Mo. ALR
Johnson, Thomas H., '23..30 W. 59th, New York 19, N. Y. OP
Johnston, Hewitt, '36...303 Exchange bldg., Orlando, Fla. ALR
Johnston, James Gilliam, '39
1520 E. Fourth, Charlotte 4, N. C. OP
Johnston, Kenneth B., '32
1509 Sherbrooke st. W., Montreal, Can. OP
①Johnston, W. H., '12..1515 State, Santa Barbara, Calif. OALR
Johnston, Wm. Raby, '42, Lt. (M.C.), U.S.N.R.,
c/o Fleet P. O., San Francisco, Calif. ALR
Joinville, Euclide V., '38...Stroh bldg., Detroit 26, Mich. ALR
Jones, Cecil Cain, '28.Equitable bldg., Des Moines 9, Iowa OALR
Jones, David E., '38
Chamber of Commerce, Indianapolis 4, Ind. ALR
Jones, David H., '35.....140 E. 54th, New York 22, N. Y. LR
Jones, Edley H., '28...1301 Washington, Vicksburg, Miss. OALR
Jones, Edmund L., '26..Wheeling Clinic, Wheeling, W. Va. OALR
Jones, Edward Earl, '40
141 West Elm st., Brockton 8, Mass. ALR
①Jones, Elgin W., '13.....44 Atlanta, Lynn, Mass. OP A
②Jones, F. W., '19.....101 E. Broadway, Alton, Ill. OALR
Jones, Florentine B., '39.....416 S. 17th, Mattoon, Ill. ALR
Jones, Fred G., '36.....258 Genesee, Utica 2, N. Y. ALR
Jones, Leonard W., '25.16 N. Goodman, Rochester 7, N. Y. OALR
Jones, Lester T., '37, Comdr. (M.C.),
U. S. Naval Base Hosp., Bremerton, Wash. ALR
Jones, M. F., '30.....121 E. 60th, New York 22, N. Y. ALR
Jones, U. G., '27....Market & Boone, Johnson City, Tenn. ALR
Jones, Vincent L., '28
Missouri Theatre bldg., St. Louis 3, Mo. OALR
Jones, W. Yerby, '43.....436 Sycamore, Buffalo 4, N. Y. OP

Jones, William S., '27
521 Sheridan road, Menominee, Mich. **OALR**
Jordan, George T., '21.30 N. Michigan av., Chicago 2, Ill. **OALR**
Jordan, James Shannon, '40
Medical Arts bldg., Scranton 3, Pa. **OP**
Jordan, Lewis W., '41. Medical Arts bldg., Portland 5, Ore. **ALR**
Jordan, Philip J., '43
910 Medico-Dental bldg., San Jose 20, Calif. **ALR**
Joy, Harold H., '26.. State Tower bldg., Syracuse 2, N. Y. **OP**
Joyce, Wm. M., '36.....121 Main st., Middleton, Conn. **ALR**
Joynt, A. J., '24.....525 Sycamore, Waterloo, Iowa **OALR**
Joynt, M. J., '30..... Royal bldg., Le Mars, Iowa **ALR**
Judd, Delbert K., '35.....950 E. 59th, Chicago 37, Ill. **ALR**
②Judd, H. B., '17.....40 E. 61st, New York 21, N. Y. **ALR**
Judd, J. H., '30..... Medical Arts bldg., Omaha 2, Nebr. **OP**
Judge, Harry V., '20.....46 Willett, Albany 6, N. Y. **OP**
Judge, Thomas A., '37...735 N. Water, Milwaukee 2, Wis. **ALR**
Juers, Arthur L., '37..... Heyburn bldg., Louisville 2, Ky. **ALR**

K

Kafka, M. Martyn, '40
109-05 72nd av., Forest Hills, L. I., N. Y. **ALR**
Kahn, Kenneth Myer, '40. Lt. Col. (M.C.), Halloran
General Hospital, Staten Island, N. Y. **ALR**
Kammerling, Theo. S., '32.616 N. Central, Chicago 44, Ill. **OALR**
Kane, J. T., '37.88-49—163rd st., Jamaica 3, L. I., N. Y. **ALR**
Kaplan, Carl, '37....69-17 Forest av., Brooklyn 27, N. Y. **ALR**
Kaplan, Leon, '42..881 Lafayette st., Bridgeport 4, Conn. **OP**
Kaplan, Samuel, '35, Major (M.C.), 0-439073
A.P.O. 813, c/o P. M., New York, N. Y. **ALR**
Kapp, Carl G., '35.440 Westminster av., Elizabeth 3, N. J. **ALR**
Kapp, Henry, '36.....50 Merrimack, Haverhill, Mass. **ALR**
Karkeet, Robert B., '28.833 S.W. 11th av., Portland 5, Ore. **ALR**
Kasnetz, Joseph P., '39
678 Eastern Parkway, Brooklyn 13, N. Y. **ALR**
Kasper, Kelvin Anthony, '38
135 S. 18th, Philadelphia 3, Pa. **ALR**
Katz, Dewey, '37.....99 Pratt, Hartford 3, Conn. **OP**
Katz, Harry Kolman, '42
1206 Cottman st., Philadelphia 11, Pa. **OP**
Katz, I. Herbert, '41, Capt. (M.C.),
A.P.O. No. 505, c/o P. M., New York **OP**
Kauffman, A. B., '22..... Chimes bldg., Syracuse 2, N. Y. **ALR**
Kauffman, M. Luther, '38
Medical Arts bldg., Jenkintown, Pa. **OP**
②Kaufman, A. Spencer, '17
1923 Spruce, Philadelphia 3, Pa. **ALR**
Kaufman, Maxwell, '40.....85 Maujer, Brooklyn 6, N. Y. **ALR**
Kaufman, Samuel I., '41.185 N. Wabash av., Chicago 1, Ill. **OP**
Kayser, Reuben, '39
442 Bay Ridge Parkway, 75th st., Brooklyn 9, N. Y. **ALR**
Kazanjian, V. H., '33
475 Commonwealth av., Boston 15, Mass. **LR-PI S**
Kearney, Harold L., '37
1403 Delachaise, New Orleans 15, La. **ALR**
Kearny, R. A., '32
2311 Connecticut av. N.W., Washington 8, D. C. **ALR**
Keating, T. F., '27..... Maccabee bldg., Detroit 2, Mich. **OALR**
Keefe, William J., '29
205 E. Sidney av., Mt. Vernon, N. Y. **OALR**
Keeler, Lansing H., '34..89-04—148th, Jamaica 2, N. Y. **OALR**
Keeler, Nelson S., '39....411 Thirtieth, Oakland 9, Calif. **ALR**
Keil, Frank C., '39..660 Madison av., New York 21, N. Y. **OALR**
Keil, Peter A., '36.118 Pennsylvania av., Brooklyn 7, N. Y. **ALR**
Keim, W. F., '24.....25 Roseville av., Newark 7, N. J. **ALR**
Keiper, G. F., Jr., '30.....113 N. Church, Visalia, Calif. **ALR**
②Keith, W. E., '16. Professional bldg., Kansas City 6, Mo. **OALR**
Keller, S. A., '20.303 S. Minnesota av., Sioux Falls, S. D. **OALR**
Kelley, Isaac D., Jr., '28
3720 Washington blvd., St. Louis 8, Mo. **ALR**
Kelley, Vincent J., '33
520 Commonwealth av., Boston 15, Mass. **ALR**
Kelly, Geo. F. J., '34.....37 S. 20th, Philadelphia 3, Pa. **OP**
Kelly, Joseph D., '28
140 E. 54th, New York 22, N. Y. **OALR-PI S**
Kemler, Joseph I., '22.1908 Eutaw pl., Baltimore 17, Md. **OALR**
Kemp, Roy C., '38.....343 State, Albany 6, N. Y. **ALR**
Kend, Lee, '32.....133 E. 58th, New York 22, N. Y. **ALR**
Kennedy, Eldred W., '28
Gas & Electric bldg., Rochester 4, N. Y. **OP**
Kennedy, Francis L., '43
Missouri Theatre bldg., St. Louis 3, Mo. **ALR**
Kennedy, Patrick James, '40
6910 Market st., Upper Darby, Pa. **OP**
Kennedy, Roscoe J., '39, Lt. Comdr. (M.C.),
3342 Sutton rd., Cleveland 20, Ohio **OP**
Kennedy, Wm. A., '36....372 St. Peter, St. Paul 2, Minn. **OP**
Kennon, W. G., '24..... Doctors' bldg., Nashville 3, Tenn. **OALR**
Kenyon, B. B., '42, Lt. Comdr. (M.C.),
N.T.S., Fort Schuyler, Bronx 61, N. Y. **ALR**
Kerns, Thomas C., '41....1110 West Main, Durham, N. C. **ALR**
Kershner, C. M., '43.... Watson Clinic, Brookings, S. Dak. **OP..**
Kershner, W. E., '34.....117 Front st., Bath, Maine **OALR**
Kerst, John Arthur, '42, Capt. (M.C.),
R.F.D. No. 4, Springfield, Ill. **OP**
Kerwin, Raymond W., '38
310 S. Michigan av., Chicago 4, Ill. **OALR**
Kessler, Mana, '38.....311 Center av., Bay City, Mich. **ALR**
Kettelkamp, Fred O., '26
Ferguson bldg., Colorado Springs, Colo. **ALR**
Key, Sam N., '26..... Norwood bldg., Austin 16, Texas **OALR**
Keyes, John E. L., '33, Major (M.C.),
Bushnell General Hospital, Brigham, Utah **OP**
Keys, Morton J., '22
Pemberton bldg., Victoria, B. C., Can. **OALR**
Keyton, John A., '34.....304 N. Oates, Dothan, Ala. **ALR**
②Kiehle, Frederick A., '15
1020 Southwest Taylor, Portland 5, Ore. **OP**
Kilbourne, P. H., '24
Fidelity Medical bldg., Dayton 2, Ohio **OALR**
Kilgore, George L., '35, Major (M.C.), A.P.O. 923,
c/o Postmaster, San Francisco, Calif. **OP**
Kimata, Harold T., '43, Lt. (M.C.), 0-372955,
A.P.O. 568, c/o Postmaster, New York, N. Y. **ALR**
Kimbrig, Max, '40.....20 High st., Huntington, N. Y. **ALR**
①Kincaid, J. H., '03
421 West Church av., Knoxville 42, Tenn. **OALR**
Kincannon, LeRoy T., '28, Major (M.C.),
216th General Hospital, Camp Forrest, Tenn. **OALR**
King, Charles R., '20..... Spitzer bldg., Toledo 4, Ohio **OALR**
King, Clair B., '37.. First Nat'l Bank bldg., Canton 2, Ohio **OP**
King, Edward, '21..... Carew Tower, Cincinnati 2, Ohio **ALR**
King, George L., Jr., '29, Lt. Col. (M.C.),
111th Evacuation Hosp., Camp Swift, Texas **OP**
King, Lowell W., '33.....470 E. State, Salem, Ohio **ALR**
③King, O. H., '16... Medical Arts bldg., Hot Springs, Ark. **OALR**
King, Walter F., '38...519 Delaware av., Buffalo 2, N. Y. **OP**
Kinkade, Joseph M., '38
359 E. Manchester blvd., Inglewood, Calif. **ALR**
Kinney, Charles Ed., '34
10515 Carnegie av., Cleveland 6, Ohio **ALR**

Kirby, D. B., '24....780 Park av., New York 21, N. Y. **OP-PI S**
Kirch, Walter, '43
417 Bankers Trust bldg., Des Moines 9, Iowa **ALR**
Kirkbright, James C., '35..40 E. 61st, New York 21, N. Y. **ALR**
Kirkpatrick, Hazen L., '37, Capt. (M.C.),
1320 Pembroke Lane, Topeka, Kansas **ALR**
③**Kirkpatrick, Henry,** '24, Lt. Col.,
54 Welbeck st., W. 1, London, Eng.
②**Kistler, O. F.,** '18...43 N. Franklin, Wilkes-Barre, Pa. **OP.AR**
②**Kistner, F. B.,** '18.....Mayer bldg., Portland 5, Ore. **ALR**
Klawans, Harold L., '43, Capt. (M.C.),
McCloskey General Hospital, Temple, Texas **ALR**
Kleinert, Margaret N., '29....1101 Beacon, Boston, Mass. **ALR**
Kleinfeld, Louis, '36.....17 E. 84th, New York 28, N. Y. **ALR**
Kler, Joseph H., '37
151 Livingston av., New Brunswick, N. J. **ALR**
Klien, Bertha A., '38
58 E. Washington, Sec. 2, Chicago 2, Ill. **OP**
①**Kline, H. G.,** '12...State Tower bldg., Syracuse 2, N. Y. **ALR**
Kline, O. R., '30.....514 Cooper, Camden, N. J. **ALR**
①**Klinedinst, John F.,** '06.....238 S. George, York, Pa. **OP A**
Klingner, Thomas O., '20
Medical Arts bldg., Springfield, Mo. **OALR**
Klotz, William F., '38, Lt. Col. (M.C.),
5701 Jackson, Apt. 1007, Houston, Texas **ALR**
Kluever, Herman C., '36, Lt. Comdr. (M.C.),
Carver bldg., Fort Dodge, Iowa **OP**
①**Knapp, Arnold,** '08.....10 E. 54th, New York 22, N. Y. **OP**
Knapp, Arthur A., '33, Comdr. (M.C.), Base Hosp. No. 4,
Navy 133, Fleet Post Office, San Francisco, Calif. **OP**
Knapp, F. N., '25....Medical Arts bldg., Duluth 2, Minn. **OP**
Knauer, Wm. J., '30...Buckman bldg., Jacksonville 2, Fla. **OALR**
Knight, Granville F., '37
Medical Centre bldg., White Plains, N. Y. **ALR**
Knight, John S., '32, Lt. Comdr. (M.C.), U.S.N.R.
162 Hillcrest Road, Berkeley 5, Calif. **ALR**
Knighton, Willis S., '31..121 E. 61st, New York 21, N. Y. **OP**
Knisely, Alan DeG., '38, Major (M.C.),
Station Hospital, Camp McCain, Miss. **ALR**
Knopf, Saul, '37.....30 E. 40th, New York 16, N. Y. **ALR**
Knowles, W. Mood, '39.Medical Arts bldg., Dallas 1, Texas **ALR**
Knowlton, Don S., '39, Capt. (M.C.), Medical Field
Service School, School Battalion Training Center,
Camp LeJeune, New River, N. C. **ALR**
Kobrak, Heinrick G., '38, Capt. (M.C.),
Fort Mason, San Francisco, Calif. **ALR**
Koch, Ferdinand L. P., '40,
780 Park av., New York 21, N. Y. **OP**
Koch, Harold J., '43..6235 S. Kedzie av., Chicago 29, Ill. **OP**
Koebbe, E. E., '29, Comdr. (M.C.),
U. S. Naval Hospital, Great Lakes, Ill. **ALR**
Koenig, Ivan J., '28.....40 W. North st., Buffalo 2, N. Y. **OP**
Kogut, Lewis V., '41, Capt. (M.C.),
A.P.O. 4834, c/o Postmaster, New York **OP**
③**Koller, Carl,** '34.....68 E. 86th, New York 28, N. Y. **OP**
Koller, H. M., '36.Medical Arts bldg., Minneapolis 2, Minn. **OP**
Koller, Louis R., '32
Medical Arts bldg., Minneapolis 2, Minn. **ALR**
①**Kopetzky, Samuel J.,** '08, Col. (M.C.),
71 East 80th, New York 21, N. Y. **ALR**
Kopp, Mortimer M., '42.79 Argyle rd., Brooklyn 18, N. Y. **ALR**
Kornberg, Maurice, '38...441 E. 15th, New York 3, N. Y. **ALR**
Kornzweig, Abraham L., '37
1200 Fifth av., New York 29, N. Y. **OP**
Kos, Clair M., '43, Major (M.C.), 0356226, School of
Aviation Medicine, Randolph Field, Texas **ALR**
①**Kraft, Oscar H.,** '12
1518 N. Dearborn parkway, Chicago 10, Ill. **OP**
Krall, J. Thomas, '42, Major (M.C.),
Station Hospital, Camp Shanks, N. Y. **OP**
Kramer, Julius C., '34.....130 E. Market, Sandusky, Ohio **ALR**
Krause, Arlington C., '37...950 E. 59th, Chicago 37, Ill. **OP**
①**Krauss, Frederick,** '10
1930 Chestnut, Philadelphia 3, Pa. **OALR**
Kravitz, Daniel, '40...861 Park place, Brooklyn 16, N. Y. **OP**
Kreag, Karl A., '39....167 Joralemon, Brooklyn 2, N. Y. **ALR**
①**Krebs, Adolph,** '13.....500 Penn av., Pittsburgh 22, Pa. **OP**
Kreman, Abraham, '41, Capt. (M.C.),
514 W. Rowland av., Covina, Calif. **OP**
②**Kress, George H.,** '18
Apt. 506, 2200 Leavenworth st., San Francisco 11, Calif. **OALR**
Krewson, William Egbert, 3rd, '40
1930 Chestnut st., Philadelphia 3, Pa. **OP**
Krieger, Alexander A., '43, Major (M.C.), Sta. Hosp.,
Indiantown Gap Military Reservation, Pa. **OP**
①**Krieger, Geo. M.,** '10,
701 Washington, Michigan City, Ind. **OALR**
②**Krieger, Wm. A.,** '19
103 Hooker av., Poughkeepsie, N. Y. **OALR**
Krimsky, Emanuel, '32, Major (M.C.),
Percy Jones Hospital, Battle Creek, Mich. **OP**
Krimsky, Joseph, '28.....Rio Road, Charlottesville, Va. **OALR**
Krone, William Frank, '42
31 Lincoln Park, Newark 2, N. J. **ALR**
Kronenberg, Bernard, '40
162 W. 54th, New York 19, N. Y. **OP**
Kronfeld, Peter C., '31.....904 W. Adams, Chicago 7, Ill. **OP**
①**Krug, Ernest F.,** '08..988 Fifth av., New York 21, N. Y. **OP**
Kruskal, I. D., '25...101 Lafayette av., Brooklyn 17, N. Y. **OP**
Krystaf, Joseph G., '37
1004 Fillmore av., Buffalo 11, N. Y. **ALR**
Kuhn, H. S., '30.....112 Rimbach, Hammond, Ind. **OP**
Kuhn, Hugh A., '28.....112 Rimbach, Hammond, Ind. **OALR**
Kully, B. M., '22.3875 Wilshire blvd., Los Angeles 5, Calif. **ALR**
Kully, Herman E., '35.Medical Arts bldg., Omaha 2, Nebr. **ALR**
Kulvin, Max M., '31.....55 E. Washington, Chicago 2, Ill. **OALR**
Kundert, Fred W., '38...The Monroe Clinic, Monroe, Wis. **ALR**
Kuritzky, Isadore, '40
1480 Nostrand av., Brooklyn 26, N. Y. **ALR**

L

Lachman, George S., '37, Lt. Comdr. (M.C.),
2201 Broadway, San Francisco 15, Calif. **OP**
LaClair, Charles Huston, Jr., '40, Lt. Col. (M.C.),
820 W. Marshall, Norristown, Pa. **ALR**
Laff, H. I., '30.....Metropolitan bldg., Denver 2, Colo. **ALR**
①**La Force, E. F.,** '08
Iowa State Bank bldg., Burlington, Iowa **OALR**
LaGattuta, Frank, '40
2488 Grand Concourse, Bronx 58, N. Y. **OP**
Laird, Archibald, '39, Lt. Col. (M.C.), 0-261916,
A.P.O. 7582, c/o P. M., New York, N. Y. **OP**
Laird, Robert G., '32.114 E. Fulton, Grand Rapids 2, Mich. **OP**
Lamb, Harry H., '29.....220 Main, Davenport, Iowa **ALR**
①**Lamb, R. S.,** '06, Stoneleigh court, Washington 6, D. C. **OP**
Lamberson, Frank Abrom, '42
19600 Grand River, Detroit 23, Mich. **ALR**
Lamberson, Wm. H., '37
1st Nat'l Bank bldg., Colorado Springs, Colo. **ALR**
Lambert, Robert K., '32,
1010 Fifth av., New York 28, N. Y. **OP**

LaMotte, Wm. O., '35
Medical Arts bldg., Wilmington 9, Del. **ALR**
Lampert, Max T., '31.....Northwest Clinic, Minot, N. D. **ALR**
①**Lancaster, W. B.**, '13
520 Commonwealth av., Boston 15, Mass. **OP**
Landegger, George P., '40, Capt. (M.C.),
401 N. Mansfield, Los Angeles 14, Calif. **OP**
Landis, J. E., '30.....232 N. Sixth, Reading, Pa. **ALR**
Lane, Franklin F., '33, Capt. (M.C.), U. S. Naval Hosp.,
Quarters "H", Chelsea 50, Mass. **OALR**
Lang, George H., '32.....202 E. Liberty, Savannah, Ga. **ALR**
Lange, Adolph C., '37.....3903 Olive, St. Louis 8, Mo. **OP**
Lapointe, Jean, '39....300 Carre St. Louis, Montreal, Can. **ALR**
LaReau, Hector G., '35..4803 Lincoln av., Chicago 25, Ill. **ALR**
③①**Large, Secord H.**, '04...170 Eddy rd., Cleveland, Ohio **ALR**
Larimore, Fred Campbell, '40
500 Penn av., Pittsburgh 22, Pa. **OP**
②**Larkin, Bernard J.**, '16
305 Hume Mansur bldg., Indianapolis 4, Ind. **OP**
③**Larsell, Olaf**, '43, Ph.D., Sc.D.,
University of Oregon, Portland, Ore.
②**Larsen, Carl L.**, '16.Lowry Med. bldg., St. Paul 2, Minn. **OALR**
Larson, Bertil T., '38.....Riker bldg., Pontiac 15, Mich. **ALR**
Larson, G. Arthur, '33.....A.O.U.W. bldg., Fargo, N. D. **OALR**
LaRue, C. L., '22.....Highland Clinic, Shreveport 50, La. **OALR**
Lasky, Mortimer A., '39, Major (M.C.), Sta. Hosp.
Army Air Base, Great Falls, Mont. **OP**
②**Lassen, Fritz**, '15....Pueblo Clinic bldg., Pueblo, Colo. **OALR**
Lassiter, Chester L., '38
Hamilton Nat'l Bk., Chattanooga 2, Tenn. **ALR**
Last, Murray A., '36...1095 Park av.,.New York 28, N. Y. **OP**
Lathbury, Edwin, '42, Major (M.C.),
Moore General Hospital, Swannanoa, N. C. **ALR**
Lathrop, Frank D., '40, Major (M.C.),
103 Moran rd., Detroit 30, Mich. **ALR**
Latimer, Marvin L., '38
129 Whitney av., New Haven 10, Conn. **ALR**
③**Lauber, Prof. Dr. Hans**, '34
Ul Gertrudy 18ms, Krakav, Generalgouvernment **OP**
Lauppe, Frederick Andrew, '40
Capt. (M.C.), 29th Field Hospital, Camp Bowie, Texas **OP**
Laval, Joseph, '31.....136 E. 64th, New York 21, N. Y. **OP**
②**La Vigne, A. A.**, '18
667 Madison av., New York 21, N. Y. **OALR**
Law, Frederick M., '37..140 E. 54th, New York 22, N. Y. **ALR**
①**Lawrence, Granville A.**, '13
Medical Arts bldg., Philadelphia 3, Pa. **OP**
Lawson, Lawrence J., '31.....636 Church, Evanston, Ill. **ALR**
Lawwill, Stewart, '28
Medical Arts bldg., Chattanooga 3, Tenn. **OALR**
Lazar, A. M., '38.....407 Eustis, Huntsville, Ala. **ALR**
Lazar, Nathan K., '36....434 Roscoe st., Chicago 13, Ill. **OP**
Leach, Robert S., '41
Medical Arts bldg., Knoxville 2, Tenn. **OP**
Leahey, Brendan D., '36.....9 Central, Lowell, Mass. **OP**
Leahey, George A., '37.....45 Merrimack, Lowell, Mass. **OP**
Leamer, Bruce V., '38, Comdr. (M.C.), Address Unknown **ALR**
Leasure, J. Kent, '31
611 Hume-Mansur bldg., Indianapolis 4, Ind. **ALR**
Leavenworth, R. O., '30
Lowry Med. bldg., St. Paul 2, Minn. **OP**
①**Leavy, C. A.**, '12...Metropolitan bldg., St. Louis 3, Mo. **ALR**
Lebensohn, James E., '27, Comdr. (M.C.),
20 E. Delaware place, Chicago 11, Ill. **OALR**
Le Compte, E. D., '27
9 Exchange pl., Salt Lake City 1, Utah **OALR**
Lederer, Francis L., '25, Comdr. (M.C.),
307 N. Michigan av., Chicago 1, Ill. **ALR**
Lee, Wayne Robert, '43
613 Medical Arts bldg., Burlington, Iowa **ALR**
Leech, V. M., '30.....55 E. Washington, Chicago 2, Ill. **OP**
LeFevre, Willis H., '35.....234 N. Duke, Lancaster, Pa. **ALR**
Lefkon, Irving M., '39..37-14 73rd, Jackson Heights, N. Y. **ALR**
②**Lefler, Anna B.**, '18
610 S. Broadway, Los Angeles 14, Calif. **OALR**
Leggett, Albert E., '34
614 Fincastle bldg., Louisville 2, Ky. **OP**
②**Lehan, James W.**, '16.....1620 12th av., Greeley, Colo. **OALR**
Lehrfeld, Louis, '39.....1321 Spruce, Philadelphia 7, Pa. **OP**
Leich, Charles F., '41, Lt. (M.C.), U.S.N.R., Office of Naval
Officer Procurement, Missouri Pacific bldg.,
St. Louis 3, Mo. **ALR**
Leinfelder, Placidus J., '37
University Hosp., Iowa City, Iowa **OP**
LeJeune, F. E., '27...Ochsner Clinic, New Orleans 15, La. **ALR**
Lemel, Samuel L., '35.1030 Euclid av., Cleveland 15, Ohio **ALR**
①**Lemore, H. B.**, '12
9615 Brighton Way, Beverly Hills, Calif. **OALR**
Lemoine, A. N., '20.....Rialto bldg., Kansas City 6, Mo. **OP**
Lempert, Julius, '43....119 E. 74th, New York 21, N. Y. **ALR**
Lennon, Robert Watson, '41.....Morris bldg., Joliet, Ill. **OP**
Lentine, Joseph, '38, Capt. (M.C.),
Lawson General Hospital, Atlanta, Ga. **ALR**
Leopold, Irving H., '43, University Hospital,
University of Pennsylvania, Philadelphia 4, Pa. **OP**
Lepard, Cecil W., '37
David Whitney bldg., Detroit 26, Mich. **OP**
Lerner, Macy L., '23.....332 Park av., Rochester 7, N. Y. **OP**
Leshin, Norman, '31.....5480 Cornell av., Chicago 15, Ill. **ALR**
①**Lester, Harry S.**, '09.....Murray bldg., Streator, Ill. **OALR**
Le Tellier, Forrest S., '36.Doctors bldg., Knoxville 30, Tenn. **OP**
Levbarg, John J., '37, Major (M.C.),
176 W. 87 st., New York 24, N. Y. **ALR**
Levin, Abram A., '37....Carew Tower, Cincinnati 2, Ohio **OP**
Levin, Nathaniel M., '39, Comdr. (M.C.),
Naval Air Station, Richmond, Fla. **ALR**
Levitt, Jesse M., '35, Capt. (M.C.),
Fletcher General Hospital, Cambridge, Ohio **OP**
Levy, Abram, '34.....1120 W. Seventh, Plainfield, N. J. **OP**
Levy, Harry H., '31.....119 Murray, Binghamton, N. Y. **OP**
Levy, Irvin, '39.....222 W. State, Trenton 8, N. J. **OP**
②**Levy, Louis**, '16..Commerce Title bldg., Memphis 3, Tenn. **ALR**
①**Levy, Robert**, '96....Metropolitan bldg., Denver 2, Colo. **ALR**
Lewin, Michael, '42, Capt. (M.C.),
Bushnell Gen. Hosp., Brigham City, Utah **ALR**
Le Win, Thurber, '29....112 Linwood av., Buffalo 9, N. Y. **OP**
②**Lewis, A. C.**, '16...130 Madison av., Memphis 3, Tenn. **OALR**
Lewis, Fielding O., '21.....(Retired), Media, Pa. **ALR**
Lewis, George A., '20.....213 Main, Roundup, Mont. **OALR**
Lewis, J. D., '21.....725 Fourth, Marysville, Calif. **OALR**
②**Lewis, James P.**, '15, Comdr. (M.C.), Worcester
Polytechnic Institute, Worcester, Mass. **ALR**
Lewis, Philip M., '28....Exchange bldg., Memphis 3, Tenn. **OP**
Lewis, Samuel D., '43.....49 Pearl, Hartford 3, Conn. **OP**
Lewis, Samuel J., '37.....S. F. C. bldg., Augusta, Ga. **ALR**
Lewy, Alfred, '42.....25 E. Washington st., Chicago 2, Ill. **ALR**
Lewy, Robert B., '39, Major (M.C.),
A.P.O. 923, c/o Postmaster, San Francisco, Calif. **ALR**
Leyda, James H., '27...Metropolitan bldg., Denver 2, Colo. **ALR**
Lierle, Dean M., '28
University Hospital, Iowa City, Iowa **ALR-PI S**
Lifschutz, Jacob, '25..310 S. Michigan av., Chicago 4, Ill. **OALR**

Liggett, Harold, '36.....133 E. 58th, New York 22, N. Y. **ALR**
Light, George, '37.....709 N. 11th, Milwaukee 3, Wis. **ALR**
Lillie, Harold I., '20.....Mayo Clinic, Rochester, Minn. **ALR**
Lillie, W. Ivan, '30.....255 S. 17th, Philadelphia 3, Pa. **OP**
Limbach, Earl Frederick, '40
30 W. 59th, Station G, New York 19, N. Y. **ALR**
Lindberg, Vernon Leslie, '42, Capt. (M.C.), Physicians' and Surgeons' bldg., Minneapolis 2, Minn. **OP**
Lindsay, John R., '36.....950 E. 59th, Chicago 37, Ill. **ALR**
Lingeman, B. N., '30...Ben Hur bldg., Crawfordsville, Ind. **ALR**
Lingeman, E. L., '29
315 Hume-Mansur bldg., Indianapolis 4, Ind. **ALR**
②**Linn, J. G.,** '19.....Jenkins Arcade, Pittsburgh 22, Pa. **OP**
Linton, Clair S., '37, Major (M.C.),
Foster Gen. Hosp., Jackson 115, Miss. **ALR**
Lippmann, Otto, '43.406 Scarbrough bldg., Austin 22, Tex. **OP**
①**Lischkoff, M. A.,** '13.....Blount bldg., Pensacola, Fla. **OALR**
④**Lister, Sir William T.,** '23
24 Devonshire pl., W. 1, London, Eng.
Litchfield, J. T., '21
Medical Arts bldg., Minneapolis 2, Minn. **OALR**
Littell, J. Jerome, '31
603 Hume-Mansur bldg., Indianapolis 4, Ind. **ALR**
Little, Milton F., '38.....49 Pearl, Hartford 3, Conn. **OP**
Littwin, Charles, '33...950 Queen Anne rd., Teaneck, N. J. **OALR**
Liva, Arcangelo, '40....5 Pangborn pl., Hackensack, N. J. **OALR**
Livingston, George S., '30
185 N. Wabash av., Chicago 1, Ill. **ALR**
Livshin, Norman, '36, Lt. Comdr. (M.C.), U.S.N.R.,
U. S. Naval Training Station, Sampson, N. Y. **ALR**
②**Lloyd, A. W.,** '15.....Lloyd bldg., Hammond, Ind. **OALR**
Lloyd, Ralph I., '25....14 Eighth av., Brooklyn 17, N. Y. **OP**
Lockwood, Chester C., '42, Capt. (M.C.),
Sta. Hosp., AAFTAC, Orlando, Fla. **OP**
Loder, L. L., '43, 506 Hackley
Union Nat'l Bank bldg., Muskegon 11, Mich. **OP**
Loeb, William J., '42, (M.C.), 0-357521,
A.P.O. 387, c/o Postmaster, New York, N. Y. **ALR**
Loewe, Walter R., '34, Comdr. (M.C.),
180-27 Tudor Road, Jamaica 3, L. I., N. Y. **OP**
Loftus, John Edward, '22
1930 Chestnut, Philadelphia 3, Pa. **ALR**
Logue, Lyle J., '31.....1304 Walker, Houston 2, Texas **ALR**
①**Lokey, H. M.,** '09....Medical Arts bldg., Atlanta 3, Ga. **OALR**
Lombardo, Melchiore, '23
142 Joralemon, Brooklyn 2, N. Y. **OP**
Long, John C., '36.....Metropolitan bldg., Denver 2, Colo. **OP**
Long, Samuel H., '39
Medical Arts bldg., Chattanooga 3, Tenn. **ALR**
Longaker, Edwin P., '43
41 E. Montgomery av., Ardmore, Pa. **ALR**
②**Loomis, E. A.,** '18
Medical Arts bldg., Minneapolis 2, Minn. **OALR**
Loomis, George L., '43...310 Choate bldg., Winona, Minn. **ALR**
Looper, Edward A., '23.104 W. Madison, Baltimore 1, Md. **OALR**
Lordan, John P., '39
1930 Wilshire blvd., Los Angeles 5, Calif. **OP**
Lore, John M., '36...325 W. End av., New York 23, N. Y. **ALR**
Lovesey, Burton E., '39
76 Bay State road, Boston 15, Mass. **ALR**
Lowenthal, Gerson, '35, Maj. (M.C.),
Ashburn General Hospital, McKinney, Texas **ALR**
Lower, Raymond A., '38, Comdr. (M.C.), U.S.N.R.,
670 Haas av., San Leandro, Calif. **ALR**
Lowry, B. W., '25.....Citizens Bank bldg., Tampa 2, Fla. **OALR**
Loy, A. W., '43.....Comdr. (M.C.), Address Unknown **OP**
Luber, Samuel, '38
1801 Eye st., N. W., Washington 6, D. C. **ALR**
Lucas, Harold R., '28
Medical-Dental bldg., Portland 5, Ore. **OALR**
Ludwig, William Henry, '40
Medical Arts bldg., Tacoma 2, Wash. **ALR**
②**Luedde, W. H.,** '16..Metropolitan bldg., St. Louis 3, Mo. **OP**
Luhr, Alfred F., '31.....40 W. North, Buffalo 2, N. Y. **OP**
Luke, William R. F., '38
Medical Arts bldg., Toronto 5, Can. **OP**
①**Lukens, Charles,** '06.....316 Michigan, Toledo 2, Ohio **OALR**
Lukens, J. A., '30.....316 Michigan, Toledo 2, Ohio **ALR**
Lukens, Robert McD., '20, Suite 1001, Medical
Arts bldg., 16th & Walnut, Philadelphia 3, Pa. **ALR**
Lundon, A. E., '27, Col.....1463 Bishop, Montreal, Can. **ALR**
Lundvick, Cyril V., '38
Medical Arts bldg., Tacoma 2, Wash. **OP**
Luongo, Romeo A., '31...2054 Locust, Philadelphia 3, Pa. **ALR**
Lupo, Carl W., '32, Col. (M.C.), 113th Station Hospital,
A.P.O. 3553, c/o Postmaster, New York, N. Y. **ALR**
Lupton, Irving M., '28.Medical Arts bldg., Portland 5, Ore. **OALR**
Lurie, M. H., '30.....483 Beacon, Boston 15, Mass. **ALR**
Lux, Paul, '25.....Bryant bldg., Kansas City 6, Mo. **ALR**
Lyle, Donald J., '27.....Carew Tower, Cincinnati 2, Ohio **OP**
Lyman, Harry W., '23.....308 N. 6th, St. Louis 1, Mo. **ALR**
Lynch, Joseph Sidney, '42, Capt. (M.C.),
A.A.F.T.T.C., Truax Field, Madison 7, Wis. **OP**
Lynch, Mercer G., '39, Major (M.C.), Quarters 113,
New Sec., Walter Reed Hospital, Washington, D. C. **ALR**
Lynch, Robert J., '31..Equitable bldg., Des Moines 9, Iowa **ALR**
Lyons, H. R., '22.....6 N. Michigan av., Chicago 2, Ill. **ALR**

Mc

McAdams, Wm. R., '33.....704 Congress, Portland 4, Me. **OP**
McAlester, Andrew W., Jr., '20
Bryant bldg., Kansas City 6, Mo. **OP**
McAlester, Andrew W., 3rd, '32, Lt. Comdr. (M.C.),
Naval General Hospital, Oakland 14, Calif. **OP**
McAlpine, Paul Theodore, '42, Capt. (M.C.),
Station Hospital, Keesler Field, Miss. **OP**
McAndrews, Leo F., '31...Mermont Plaza, Bryn Mawr, Pa. **OP**
McAskill, James E., '32
Woolworth bldg., Watertown, N. Y. **ALR**
McAuley, Albert G., '38
1301 Dorchester st. W., Montreal, Can. **OP**
McAuliffe, G. W., '32
630 Park av., New York 21, N. Y. **ALR-PI S**
McBean, George M., '20..25 E. Washington, Chicago 2, Ill. **OALR**
McBean, James Blish, '38, Major (M.C.),
Station Hospital, Fort Sill, Okla. **ALR**
McBride, Linn Frederick, '42
122 S. Michigan av., Chicago 3, Ill. **ALR**
McBride, Noel S., '40....12 S. 7th st., Terre Haute, Ind. **OP**
McCall, J. W., '43.....610 Rose bldg., Cleveland 15, Ohio **ALR**
McCannel, Archie D., '22
Corner 1st st. S.W. and Central av., Minot, N. D. **OALR**
McCart, Howard W. D., '39
170 St. George st., Toronto, Ontario, Can. **ALR**
McCarthy, C. T., '40.....1810 Pine, Philadelphia 3, Pa. **ALR**
①**McCaskey, Carl H.,** '11
Guaranty bldg., Indianapolis 4, Ind. **ALR**
McCaslin, Murray F., '40
Union Trust bldg., Pittsburgh 19, Pa. **OP**
McCaw, John A., '19.....Majestic bldg., Denver 2, Colo. **OP**
McClanahan, Robert, '37..60 E. 75th, New York 21, N. Y. **OP**

McClintock, Walter L., '39, Capt. (M.C.), Address Unknown **ALR**

McCloskey, Paul, '40.....338 Locust st., Johnstown, Pa. **ALR**

McClure, George, '39.....411 Thirtieth, Oakland 9, Calif. **ALR**

McCollom, Robert L., '42, Major (M.C.), 397th Bomb Group, Hunter Field, Savannah, Ga. **ALR**

McCollough, T. B., '32
121 University Place, Pittsburgh 13, Pa. **ALR**

McComiskey, Arthur James, '40
3420 Prytania st., New Orleans 15, La. **ALR**

McConkie, Willis L., '36.....Carroll, Iowa **ALR**

①**McCool, J. L.**, '11...450 Sutter, San Francisco 8, Calif. **OP**

McCormick, T. F., '30.....Wells bldg., Milwaukee 2, Wis. **ALR**

McCoy, L. L., '30.....1317 Marion, Seattle 4, Wash. **OP**

①**McCready, J. Homer**, '12
121 University pl., Pittsburgh 13, Pa. **ALR**

McCuaig, Duncan Roderick, '40, Major (M.C.), 0-413966, A.P.O. 511, c/o Postmaster, New York, N. Y. **ALR**

McCuaig, Victor C., '39...40 E. 61st, New York 21, N. Y. **ALR**

McCullock, Robert J. P., '20
Medical Arts bldg., Toronto 5, Can. **OALR**

McCullough, C. J., '29
Washington Trust bldg., Washington, Pa. **ALR**

McCullough, Lester E., '42
1553 Woodward av., Detroit 26, Mich. **OP**

McCullough, Malcolm K., '39
Medical Arts bldg., Dallas 1, Texas **OP**

McCurdy, Gordon Judson, '40
198 Angell st., Providence 6, R. I. **ALR**

McCutchan, Frank, '38....100 W. Innes, Salisbury, N. C. **ALR**

McDannald, C. E., '27...100 W. 59th, New York 22, N. Y. **OP**

McDavitt, Bertha S. S., '29..Main & Av. A, Temple, Texas **ALR**

McDermott, John J., '24..505 Pleasant, St. Joseph, Mich. **OALR**

②**McDiarmid, H. Oliver**, '18
Clement block, Brandon, Man., Can. **OALR**

②**McDonald, C. L.**, '19.....Rose bldg., Cleveland 15, Ohio **OALR**

McDonald, P. Robb, '40, Major (M.C.),
255 S. 17th, Philadelphia 3, Pa. **OP**

McDougall, J. Calhoun, '32
Medical Arts bldg., Atlanta 3, Ga. **ALR**

McDougall, Wm. L., '31....Doctors bldg., Atlanta 3, Ga. **ALR**

①**McDowell, N. D.**, '14,275 Alexander, Rochester 7, N. Y. **OALR**

McEachern, Margaret A., '39
Medical Arts bldg., Toronto, Can. **ALR**

McFarlane, Donald C., '37
380 Queens av., London, Ont., Can. **ALR**

McGannon, Francis Lambert, '41
14900 Detroit av., Lakewood 7, Ohio **ALR**

McGarry, H. Isabelle, '43.....636 Church, Evanston, Ill. **OP**

McGarvey, Wm. E., '29
Jackson City Bank bldg., Jackson, Mich. **OALR**

McGavic, John Samuel, '40
635 W. 165th, New York 32, N. Y. **OP**

McGeary, George E., '37
Medical Arts bldg., Minneapolis 2, Minn. **ALR**

McGehee, Melvin Wood, '42
Laird Memorial Clinic, Montgomery, W. Va. **ALR**

McGillicuddy, O. B., '30, Major (M.C.),
Station Hospital, Amarillo Field, Texas **ALR**

McGovern, Francis H., '39
Masonic Temple bldg., Danville, Va. **ALR**

McGowan, Joseph Francis, '43
New Medical bldg., Asheville, N. C. **ALR**

McGrath, Harry, '41
12 Shore Lane, Bay Shore, L. I., N. Y. **OP**

McGrath, James B., '37
2615 Grand Concourse, New York 58, N. Y. **ALR**

McGrath, Philip R., '39, Lt. Comdr. (M.C.),
U. S. Naval Hospital, New Orleans 12, La. **ALR**

McGregor, D'Arcy, '36.....83 Bryant, Buffalo 9, N. Y. **ALR**

McGregor, Gregor, '31....170 St. George, Toronto 5, Can. **ALR**

①**McGuire, H. H.**, '13..105 N. Braddock, Winchester, Va. **OP**

McGuire, John, '43.....Peery bldg., Bluefield, W. Va. **ALR**

McGuire, William P., '40, Lt. Comdr. (M.C.),
717 S. Stewart, Winchester, Va. **OP**

McHenry, L. Chester, '30
Medical Arts bldg., Oklahoma City 2, Okla. **ALR**

McIntire, Frederic J., '37....63 N. Common, Lynn, Mass. **ALR**

McIntyre, George F., '37.25 E. Washington, Chicago 2, Ill. **ALR**

McKay, Edward D., '43, Major (M.C.),
Brooke General Hospital, Fort Sam Houston, Texas **OP**

②**McKee, Claude W.**, '17...Coulter bldg., Greensburg, Pa. **OALR**

McKee, Joseph W., '28
Professional bldg., Kansas City 6, Mo. **OP**

McKee, Thomas L., '39, Major (M.C.),
A.P.O. 928, c/o Postmaster, San Francisco, Calif. **OP**

McKee, Thomas P., '39
Jones Hospital, Johnson City, Tenn. **ALR**

②**McKennan, James W.**, '17
Washington Trust bldg., Washington, Pa. **OP**

McKenzie, William Raymond, '41
101 W. Read st., Baltimore 1, Md. **ALR**

McKeown, Elmer E., '21...Republic bldg., Denver 2, Colo. **OALR**

McKeown, Hugh S., '31.635 W. 165th, New York 32, N. Y. **OP**
Also 9 Tanglewylde av., Bronxville, N. Y.

McKinney, A. R., '23
330 S. Washington av., Saginaw, Mich. **OP**

McKinney, James Wesley, '41
Exchange bldg., Memphis 3, Tenn. **OP**

McLane, Jesse Newman, '42..Blount bldg., Pensacola, Fla. **ALR**

McLaughlin, John James, '41, Lt. (M.C.),
U. S. Naval Training Station, Sampson, N. Y. **ALR**

McLaughlin, R. C., '43
3780 Wilshire bldg., Los Angeles 5, Calif. **ALR**

McLaurin, James Walter, '41.251 3rd, Baton Rouge 6, La. **ALR**

McLaurin, John G., '26..4710 Munger av., Dallas 4, Texas **OALR**

McLean, John M., '40...525 E. 68th, New York 21, N. Y. **OP**

McLellan, Thomas G., '35
First Nat'l Bank bldg., Connellsville, Pa. **OP**

McLemore, Carl Sloan, '40, Capt. (M.C.), Dale Mabry Field.
Station Hospital, Tallahassee, Fla. **ALR**

McLeod, Angus M., '36, Capt. (M.C.),
Medical Arts bldg., Toronto 5, Can. **ALR**

McLeod, John, '39, Major (M.C.),
Cantonment Hospital, Fort Sill, Okla. **OP**

McMahon, Bernard J., '26
Missouri Theatre bldg., St. Louis 3, Mo. **ALR**

①**McMurray, J. B.**, '13
Washington Trust bldg., Washington, Pa. **OALR**

McMurray, John S., '43, Capt. (M.C.),
Station Hospital, C.A.A.B., Charleston, S. C. **ALR**

McNair, Sterling S., '41, Lt. Comdr. (M.C.),
234 E. Vanderbilt, Corpus Christi, Texas **OP**

McNally, Wm. James, '28....Linton Apts., Montreal, Can. **ALR**

McNay, Miller Ost, '41
472 Central av., St. Petersburg 5, Fla. **ALR**

McNichols, W. A., '25.....101 W. First st., Dixon 3, Ill. **OALR**

McPherson, Samuel D., '35
McPherson Hospital, Durham, N. C. **ALR**

McQuiston, Ralph J., '37, Lt. Col. (M.C.),
203 Barter av., Kirkwood, Mo. **ALR**

McQuoid, Robert Moffat, '40....39 Columbia, Bangor, Me. **ALR**

McRae, John H., '30
Medical Arts bldg., Grand Rapids 2, Mich. **OP**

McReynolds, George S., Jr., '40
927 Strand, Galveston, Texas ALR

McRoberts, Earl S., '38..55 E. Washington, Chicago 2, Ill. ALR

McWilliams, Charles A., '27
1300 27th av., Gulfport, Miss. ALR

McWilliams, Charles S., '37, Maj. (M.C.)
205 Liberty, Newburg, N. Y. OP

M

Mabee, Oscar Judson, '41
Medical Arts bldg., Mitchell, S. D. ALR

MacCardell, Frank Cameron, '40
193 Waterman st., Providence 6, R. I. ALR

MacCready, Paul B., '31.442 Temple, New Haven 11, Conn. ALR

Macdonald, James Orville, '41
67 E. 59th, New York 22, N. Y. ALR

Macdonald, J. C., '30
301 N.W. 12th, Oklahoma City 3, Okla. ALR

MacDonald, Philip G., '35.115 E. 61st, New York 21, N.Y. ALR

Macdonald, Roderick, '40.330 East Main, Rock Hill, S. C. ALR

MacFarlane, Howard W., '41
David Whitney bldg., Detroit 26, Mich. ALR

②Macfarlane, P. B., '16
Medical Arts bldg., Hamilton, Ont., Can. OALR

①Mackenzie, George W., '12
269 S. 19th, Philadelphia 3, Pa. OALR

Mackenzie, Roland C., '39....30 Grant, Waltham 54, Mass. OP

②MacLachlan, A. A., '18..May bldg., Pittsburgh 22, Pa. ALR

MacLean, Angus L., '35.1201 N. Calvert, Baltimore 2, Md. OP

MacMillan, Andrew L., Jr., '37
46 Pleasant, Concord, N. H. ALR

MacMillan, J. A., '25.....1410 Stanley, Montreal, Can. OP

Macneil, Frank A., '38.Medical Arts bldg., Winnipeg, Can. ALR

Macnie, John P., '35, Lt. Comdr. (M.C.),
U. S. Naval Hospital, Annapolis, Md. OP

②Macnie, J. S., '19
Medical Arts bldg., Minneapolis 2, Minn. OALR

Macpherson, Duncan, '36
920 Fifth av., New York 21, N. Y. ALR

Macpherson, Wm. A., '43, Capt. (M.C.), 205 Hosp.
Complement, c/o Sta. Hosp. Camp Torrence, Calif. OP

Madeley, Howard Randall, '40
727 Sonoma st., Vallejo, Calif. OP

Magee, F. Earle, '37.....222 Seneca, Oil City, Pa. ALR

Maggard, D. I., '20.....Orpheum bldg., Wichita 2, Kan. OALR

Maghy, Charles A., '26
Wilshire Med. bldg., Los Angeles 5, Calif. OP

Mahoney, Charles L. '40, Major (M.C.), 0468652,
A.P.O. 645, c/o Postmaster, New York, N. Y. ALR

Mahoney, Frank A., Jr., '43, Capt. (M.C.), 0-350306,
A.P.O. 813, c/o Postmaster, New York, N. Y. ALR

Mahoney, Paul L., '28...Donaghey bldg., Little Rock, Ark. ALR

②Maiden, Sydner D., '18
Bennett bldg., Council Bluffs, Iowa OALR

Maier, Marcus M., '43.....513 Front, Hempstead, N. Y. ALR

Mallniac, Jacques W., '36
106 E. 78th, New York 21, N. Y. ALR-PI S

Mallon, Frank E., '37.1135 Park place, Brooklyn 13, N. Y. OP

Malone, F. B., '30...2609 Nineteenth st., Lubbock, Texas ALR

Malone, Phocian W., '36.....811 Main, Big Spring, Texas ALR

Maloy, Wayland H., '33
608½ W. Sheridan av., Shenandoah, Iowa OP

Mandeville, Clifford Bagley, '41
Hackley Union Bank, Muskegon, Mich. ALR

Maness, Guy Milford, '41
119 7th av. North, Nashville 3, Tenn. ALR

Manigan, Thomas P., '39, Major (M.C.),
Hendricks Field, Sebring, Fla. ALR

Manion, Marlow W., '36
601 Hume-Mansur bldg., Indianapolis 4, Ind. ALR

Mann, Albert H., '35
2324 Glendale av., Texarkana, Ark.-Texas ALR

Mann, B. H., '30.....1930 Chestnut, Philadelphia 3, Pa. OP

Mann, J. J., '34.....255 State, Perth Amboy, N. J. ALR

Mann, Mortimer, '43
323 Hume-Mansur bldg., Indianapolis 4, Ind. OP

Mann, William A., '38..30 N. Michigan av., Chicago 2, Ill. OP

March, Clara A., '22....465 Ashland av., Buffalo 13, N. Y. OP

Marchman, Oscar M., '35..5328 Live Oak, Dallas 6, Texas ALR

Marcotte, Raymond H., '32.....196 Main, Nashua, N. H. ALR

Marcove, Maurice E., '39...1612 Tremont, Denver 2, Colo. OP

②Markel, J. Clyde, '17
806 Pitt Bank bldg., Pittsburgh 22, Pa. OP

Marks, Julian B., '29
1093 Broxton av., Los Angeles 24, Calif. ALR

Marks, Moses Isaac, '42, Capt. (M.C.),
Newport Army Air Field, Newport, Ark. ALR

Marks, Paul L., '42, Capt. (M.C.), 0-449825,
APO 510, c/o P.M., New York, N. Y. OP

Marks, Roland F., '39, Major (M.C.),
A.P.O. 953, c/o Postmaster, San Francisco, Calif. ALR

Marks, Samuel B., '28.....200 W. 2nd, Lexington 15, Ky. ALR

Marmelstein, Maurice, '43
1st Nat'l Bank bldg., Carbondale, Pa. OP

Marsden, W. C., '30....99 N. Franklin, Wilkes-Barre, Pa. OALR

Marshall, Don, '36, Major (M.C.), 0-480194,
APO 508, c/o P.M., New York, N. Y. OP

Marshall, George G., '23.....Gryphon bldg., Rutland, Vt. OALR

Marshall, Hal E., '35...105 S. Broadway, Wichita 2, Kan. ALR

①Marshall, Watson, '14
121 University pl., Pittsburgh 13, Pa. ALR

Marshall, William J., '38..Montana block, Missoula, Mont. ALR

Martin, Ashby G., '36
895 Lexington av., New York 21, N. Y. ALR

Martin, Henry F., '40, Major (M.C.),
Station Hospital, West Point, N. Y. ALR

Martin, Hilmar G., '39
231 W. Wisconsin av., Milwaukee 3, Wis. OP

Martin, Norvil A., '37, Major (M.C.),
A.P.O. 923, c/o Postmaster, San Francisco, Calif. ALR

Martin, Paul T., '38.235 E. Santa Clara, San Jose 20, Calif. ALR

Martin, Roy D., '29....Masonic bldg., Grand Island, Nebr. ALR

Martin, W. O., Jr., '30.....478 Peachtree, Atlanta 3, Ga. OP

Mason, Roy E., '28.....Frisco bldg., St. Louis 1, Mo. OP

Masor, Philip L., '41, Capt. (M.C.),
145th Station Hospital, Fort Dix, N. J. OP

Master, John R., '43, Lt. (M.C.), Letterman
General Hospital, San Francisco, Calif. ALR

Masters, John M., '38, Major (M.C.), Ashford General
Hospital, White Sulphur Springs, W. Va. OP

Masters, Robert J., '28
805 Hume-Mansur bldg., Indianapolis 4, Ind. OP

Mather, Homer Raymond, '42.....224 Depot, Latrobe, Pa. ALR

Matheson, John H., '39
Equitable bldg., Des Moines 9, Iowa OP

Mathews, Raymond H., '38
186 South st., Morristown, N. J. ALR

Mathias, Daniel Francis, '40
Central Tower bldg., Akron 8, Ohio ALR

Matthews, John L., '43, Lt. Col. (M.C.), School of
Aviation Medicine, Randolph Field, Texas OP

①**Matthews, Justus,** '10
Medical Arts bldg., Minneapolis 2, Minn. **LR**

Mattis, Robert Dean, '42, Capt. (M.C.),
375 North Taylor av., St. Louis 8, Mo. **OP**

Matusow, A. P., '35
1100 Grand Concourse, New York 56, N. Y. **ALR**

Matzkin, Lloyd L., '43, Capt. (M.C.),
APO 929, c/o P.M., San Francisco, Calif. **ALR**

Maumenee, Alfred E., '43
Johns Hopkins Hospital, Baltimore 5, Md. **OP**

Maw, Raymond B., '39
699 E. South Temple, Salt Lake City 2, Utah **ALR**

Maxwell, Hal W., '43, Lt. (M.C.),.....Myra, Texas **OP**

Maxwell, Harvey C., '35, Lt. Col. (M.C.),
1718 N. Main, Santa Ana, Calif. **OALR**

Maxwell, James H., '34
1st Nat'l Bank bldg. Ann Arbor, Mich. **ALR**

Maxwell, Joseph S., '39
Professional bldg., Fairmont W. Va. **OP**

May, Frederick T., '41, Major (M.C.),
Deshon General Hospital, Butler, Pa. **OP**

②**May, J. V.,** '15.....1703 Main, Marinette, Wis. **OALR**

②**Maybaum, J. L.,** '16..1070 Park av., New York 28, N. Y. **ALR**

Mayer, Leo L., '28
Suite 204, 415 N. Camden drive, Beverly Hills, Calif. **OP**

Mayerberg, Emil R., '29
Medical Arts bldg., Wilmington 9, Del. **ALR**

Mead, Ernest Charles, '41
106½ E. California, Gainesville, Texas **ALR**

Means, R. G., '30.....40 S. 3rd, Columbus 15, Ohio **ALR**

Meek, Raymond E., '31, Comdr. (M.C.),
U. S. Naval Hospital, Newport, R. I. **OP**

Meinberg, William H., '39.....906 Olive, St. Louis 1, Mo. **OP**

Mellinger, Wm. J., '27
323 E. Sola st., Santa Barbara, Calif. **ALR**

Meloy, Wm. C., '33, Lt. Comdr. (M.C.,)
2019 R st. N.W., Washington 9, D.C. **ALR-PI S**

Melton, Bernard L., Jr., '31..15 E. Monroe, Phoenix, Ariz. **OP**

Meltzer, Philip E., '34.20 Charlesgate W., Boston 15, Mass. **ALR**

Mendel, James H., '32....Ingraham bldg., Miami 32, Fla. **ALR**

Mengel, Sterling Frederic, '40, Lt. Comdr. (M.C.),
612 W. Market st., Pottsville, Pa. **OP**

Menger, Louis C., '29...202 Weirfield, Brooklyn 21, N. Y. **ALR**

Mensher, Ira Wadsworth, '42.50 Plaza, Brooklyn 17, N. Y. **OP**

Menzies, Clifford G., '39
733 Woodward av., Iron Mountain, Mich. **ALR**

Mercer, Ray, '20, Lt. Col. (M.C.), P.W.I.C. Hospital,
Crossville, Tenn. **OALR**

①**Mercer, Richard E.,** '03...126 Monterey, Detroit 3, Mich. **LR**

Meredith, Lawrence Cornelius, '40
124 Middle av., Elyria, Ohio **ALR**

Merica, F. W., '30...14805 Detroit av., Lakewood 7, Ohio **ALR**

Merkel, Byron M., '38, Major (M.C.),
802 Equitable bldg., Des Moines 9, Iowa **ALR**

Merkel, C. C., '28
85 Kercheval av., Grosse Pointe Farms 30, Mich. **ALR**

Merrick, Charles Gordon, '40...Leon bldg., Ft. Myers, Fla. **ALR**

②**Merrill, H. G.,** '19..3245 Fourth av., San Diego 3, Calif. **OP**

Mertins, Paul S., Jr., '37, Capt. (M.C.),
A.A.F.A.F.S., Del Rio, Texas **ALR**

Merz, Earl H., '43, Capt. (M.C.), 0491970, Amarillo
Army Air Field, Amarillo, Texas **OP**

Mesirow, Maurice E., '35.117 E. Cook, Santa Maria, Calif. **OP**

Messenger, Harry K., '39
313 Commonwealth av., Boston 15, Mass. **OP**

①**Messinger, H. C.,** '14....210 Angell, Providence 6, R. I. **OP**

Messier, A. Eugene, '25.....390 Main, Worcester 8, Mass. **OALR**

①**Metz, Roy B.,** '12...1422 Euclid av., Cleveland 15, Ohio **OP**

Meyer, George P., '37.....410 Haddon av., Camden, N. J. **OP**

Meyer, Samuel J., '28...58 E. Washington, Chicago 2, Ill. **OP**

Meyers, Paul Thomas, '42, Major (M.C.),
82nd General Hospital, Spokane, Wash. **ALR**

Mezz, David, '37...330 New York av., Brooklyn 13, N. Y. **ALR**

①**Middleton, A. B.,** '11.....Pontiac, Ill. **OALR**

Middleton, Harry E., '20.....206A W. 3rd, Alton, Ill. **OALR**

Miksch, Henry Fred, '41.....209 S. Park, Rochester, Pa. **ALR**

Milder, Benjamin, '43.501 Humboldt bldg., St. Louis 3, Mo. **OP**

Miller, A. Boyd, '43
1006 Professional bldg., 429 Penn av., Pittsburgh 22, Pa. **ALR**

Miller, Alden H., '39, Lt. (M.C.), U.S.N.R.,
U. S. Naval Hospital, Long Beach, Calif. **ALR**

②**Miller, Alice R.,** '19
505 Guaranty bldg., West Palm Beach, Fla. **OALR**

Miller, Alvin R., '39.....4th and Pike, Seattle 1, Wash. **ALR**

Miller, Edwin Ephron, '42
Medical Arts bldg., Knoxville 1, Tenn. **ALR**

Miller, G. Leslie, '35.14804 Detroit av., Lakewood 7, Ohio **OP**

Miller, H. Edward, '30.634 N. Grand blvd., St. Louis 3, Mo. **ALR**

Miller, Hugh, '20.....Argyle bldg., Kansas City 6, Mo. **OALR**

Miller, Isidore, '37
2021 Grand Concourse, New York 53, N. Y. **ALR**

Miller, John, '30.....63 North st., Greenwich, Conn. **ALR**

Miller, Joseph A., '32...364 Prospect, South Orange, N. J. **ALR**

Miller, J. W., '29.....242 E. 19th, New York 3, N. Y. **ALR**

Miller, M. Valentine, '34
114 W. Phil-Ellena, Mt. Airy, Philadelphia 19, Pa. **ALR**

Miller, Ralph H., '39, Capt. (M.C.),
6257 Rogers Park pl., Pleasant Ridge, Cincinnati 13, Ohio **OP**

Miller, Saul, '34.....88-05 150th st., Jamaica 2, N. Y. **OP**

Miller, Sidney N., '43
113 Hooker av., Poughkeepsie, N. Y. **ALR**

Miller, Theo. E., '31..First Trust bldg., Pasadena 1, Calif. **ALR**

Millett, Frank A., '33.....40 High, Greenfield, Mass. **OALR**

Milligan, Roy H., '36..3720 Washington, St. Louis 8, Mo. **ALR**

Mills, Chester R., '34
520 Commonwealth av., Boston 15, Mass. **ALR**

Minns, John Edwards, '41....316 Michigan, Toledo 2, Ohio **ALR**

②**Minor, H. F.,** '18
Commerce Title bldg., Memphis 3, Tenn. **OALR**

Minsky, Arthur, '43..8601 Homelawn st., Jamaica 3, N. Y. **OP**

Minsky, Henry, '26.....2 E. 95th, New York 28, N. Y. **OP**

Minton, Wm. H., '20.....Corby bldg., St. Joseph 9, Mo. **OALR**

Mirow, David, '38.....237 E. 20th, New York 3, N. Y. **ALR**

Mishler, Donald L., '36
604 S. Cincinnati av., Tulsa 3, Okla. **ALR**

②**Mitchell, E. K.,** '18
710 W. Lehigh av., Philadelphia 33, Pa. **ALR**

Mitchell, H. E., '30..14805 Detroit av., Cleveland 7, Ohio **ALR**

①**Mithoefer, W.,** '11.....Carew Tower, Cincinnati 2, Ohio **ALR**

①**Mittendorf, A. D.,** '12.399 Park av., New York 22, N. Y. **OP**

Mobley, Marion R., '39, Lt. Col. (M.C.),
Deshon General Hospital, Butler, Pa. **ALR**

Moehle, Walter, '32...15 Schermerhorn, Brooklyn 2, N. Y. **OP**

Moffat, Harold Forster, '42, The Clinic,
South Hotel st., at Thomas sq., Honolulu, Hawaii **OP**

Moffett, Daniel B., '38
1150 Connecticut av. N.W., Washington 6, D. C. **ALR**

Mohun, Meade, '43.....23 Second av., San Mateo, Calif. **ALR**

Moll, Arthur M., '32
Powers Theatre bldg., Grand Rapids 2, Mich. **ALR**

Molsberry, Jasper Merl, '41
425 E. Wisconsin av., Milwaukee 2, Wis. **OP**

①Molt, Wm. F., '11
Bankers Trust bldg., Indianapolis 4, Ind. ALR
Monaco, Louis, '43
233 S. San Fernando blvd., Burbank, Calif. ALR
①Monahan, J. A., '13.....181 Chestnut, Clinton, Mass. OALR
Monahan, James J., '35.....31 S. Jordin, Shenandoah, Pa. ALR
Moncreiff, William F., '38.58 E. Washington, Chicago 2, Ill. OP
Mond, Edward, '40.....Eaton Tower, Detroit 26, Mich. ALR
Monette, C. J., '37.....814 Elm, Manchester, N. H. ALR
①Monosmith, O. B., '05.....O. P. S. bldg., Lorain, Ohio OALR
Monserrate, Manuel Maria, '41
73 Main, Binghamton, N. Y. ALR
Monson, LaFayette, '34.450 Sutter, San Francisco 8, Calif. ALR
①Monson, S. H., '10.B. F. Keith bldg., Cleveland 15, Ohio OALR
Montgomery, D. Cameron, '28
Weinberg bldg., Greenville, Miss. ALR
Montgomery, John Lee, '40, Major (M.C.),
Station Hospital, Sheppard Field, Texas OP
Moon, Seymour B., '36
429 Pennsylvania av., Pittsburgh 22, Pa. OP
Moore, Donald E., '43, Lt. Comdr. (M.C.), U.S.N.,
1100 Euclid av., Syracuse 10, N. Y. OP
Moore, Ellis Columbus, '41
First National Bank bldg., Olean, N. Y. ALR
②Moore, G. A., '16.....Box 471, Palmer, Mass. OALR
②Moore, George H., '19.101 Long Lane, Upper Darby, Pa. OALR
Moore, J. Hallock, '35.1050 Fifth av., Huntington 1, W.Va. ALR
Moore, James A., '41, Major (M.C.), 0-439771,
A.P.O. 928-1, San Francisco, Calif. ALR
Moore, P. G., '30....Republic bldg., Cleveland 15, Ohio OP
Moore, Paul M., Jr., '30.Cleveland Clinic, Cleveland 6, Ohio ALR
Moore, R. B., '30.....2000 Main, Little Rock, Ark. ALR
Moore, Roy S., '28...State Tower bldg., Syracuse 2, N. Y. ALR
①Moore, T. W., '01..1050 Fifth av., Huntington 1, W. Va. OP
Moore, Thomas Scott, '39.....107 N. 2nd, Niles, Mich. OP
Moore, Walter V., '37....1 Nevins st., Brooklyn 17, N. Y. OP
Moorhead, Robert L., '33..125 Remsen, Brooklyn 2, N. Y. ALR
Moran, Charles T., '37, Maj. (M.C.), 0-481069,
Station Hospital, Kelly Field, Texas OP
Morgan, Alonzo Lloyd, '40.170 St. George, Toronto 5, Can. OP
Morgan, Harold Eugene, '42....210 S. Pine, Newton, Kan. OP
Morgan, Irving J., '39
Jenkins Arcade bldg., Pittsburgh 22, Pa. OP
Morgan, William A., '39
1835 Eye st. N.W., Washington 6, D. C. ALR
Morgana, Dante James, '41.....13 Main, Lockport, N. Y. OP
Moriarty, Richard W., '28
3 Mamaroneck Road, White Plains, N. Y. OALR
Moriconi, Albert F., '39, Major (M.C.),
104 E. Ferry Road, Morrisville, Pa. ALR
Morledge, Roy V., '33....Hart-Albin bldg., Billings, Mont. ALR
Morley, George A., '30....P.O. Box 452, Crookston, Minn. OP
Morman, William Daniel, '41
3507 Franklin av., St. Louis 6, Mo. ALR
②Morris, A. G., '18.Medical Arts bldg., Rochester 7, N. Y. ALR
Morris, Samuel Archard, '41, Capt. (M.C.), Station
Hospital, Camp Anza, Arlington, Calif. OP
Morrison, Frederick M., '37, Lt. Comdr. (M.C.),
Allied Arts bldg., Lynchburg, Va. OP
Morrison, James D., '43, Capt. (M.C.), 0-269342,
A.P.O. 502, c/o P. M., San Francisco, Calif. OP
Morrison, Wm. Howard, '38
Medical Arts bldg., Omaha 2, Nebr. OP
Morrison, W. R., '38....Hart-Albin bldg., Billings, Mont. OALR
Morrison, William W., '26, Comdr. (M.C.), Naval
Dispensary, Navy Dept., Washington 25, D. C. ALR
Morrissett, Leslie E., '37, Major (M.C.),
Borden General Hospital, Chickasha, Okla. ALR
Morrone, Francis X., '43.117 S. 2nd av., Mt. Vernon, N. Y. ALR
Morsman, L. Wm., '20....Morsman Clinic, Hibbing, Minn. OP
Morton, J. B., '24.....90 E. Warren av., Detroit 1, Mich. OALR
Morwitz, Samuel M., '36.55 E. Washington, Chicago 2, Ill. ALR
Moser, George P., '43, Capt. (M.C.),
76 Willow, Carlisle, Pa. ALR
②Mosher, Harris P., '18....127 Front, Marblehead, Mass. ALR
Mosher, Henry A., '42, Capt. (M.C.),
Winter General Hospital, Topeka, Kan. OP
Moshinsky, Alex. L., '36..237 E. 20th, New York 3, N. Y. ALR
Moss, L. Connor, '37, Comdr. (M.C.),
2223 Observatory place N.W., Washington 7, D. C. OP
Moss, Olin W., '31.....Weber bldg., Lake Charles, La. ALR
Motley, Fred Elliot, '38...106 W. 7th, Charlotte 2, N. C. ALR
Mott, Walter Charles, '40, Major (M.C.),
103 Lenox av., Albany 3, N. Y. OP
Motto, M. Paul, '29.....Rose bldg., Cleveland 15, Ohio OP
②Moulton, Everett C., '15
Merchants Nat'l Bk. bldg., Ft. Smith, Ark. OALR
①Moulton, H., 1900
Merchants Nat'l Bk. bldg., Ft. Smith, Ark. OALR
Mountain, Edward R., '42, Capt. (M.C.), Valley Forge
General Hospital, Phoenixville, Pa. ALR
Mowry, Ben, '40.....First Nat'l Bk. bldg., Findlay, Ohio ALR
Moyer, J. Sanford, '34..410 Park av., New York 22, N. Y. ALR
Moyle, Eugene H., '31, Lt. Comdr. (M.C.),
U. S. Naval Hospital, Newport, R. I. ALR
Mueller, Lawrence W., '39, Major (M.C.),
LaGarde General Hospital, New Orleans 12, La. ALR
Mueller, Werner, '37.....412 Beacon, Boston 15, Mass. ALR
Muldoon, W. E., '36
Nix Professional bldg., San Antonio 5, Texas OP
Mullen, Carroll R., '37...2025 Locust, Philadelphia 3, Pa. OP
Mullen, C. J., '35, Major (M.C.),
A.A.B. Hospital, Ft. George Wright, Wash. OP
Mulligan, Harold R., '37, Comdr. (M.C.), 2028 S. Pacific
Oceanside, Calif. ALR
Mulsow, J. E., '21, Lt. Comdr. (M.C.), U.S.N.R.,
1818-6th av., San Diego 1, Calif. OALR
Mumma, Claude S., '32
U. S. Veterans' Facility, Los Angeles 25, Calif. ALR
①Muncy, Wm. M., '14....162 Angell, Providence 6, R. I. OALR
①Mundt, G. Henry, '12
30 N. Michigan av., Chicago 2, Ill. OALR
Munson, F. T., '30.2201 E. Jefferson av., Detroit 7, Mich. ALR
Murphy, A. B., '30
Medical & Dental bldg., Everett, Wash. ALR
Murphy, Edward S., '36, Col. (M.C.),
309 Eddy av., Missoula, Mont. OP
Murphy, Rex L., '37...Metropolitan bldg., Denver 2, Colo. ALR
Murphy, Wm. E., '34.....390 Main, Worcester 8, Mass. ALR
Murray, Henry J., '31.....53 South st., Stamford, Conn. ALR
Murtagh, John Anthony, '41
Hitchcock Clinic, Hanover, N. H. ALR
Muskat, Irving, '33.....25 E. Washington, Chicago 2, Ill. OALR
Mussun, W. G., '24.....Rose bldg., Cleveland 15, Ohio ALR
Myers, David, '36, Major (M.C.), 0-1696046, A.P.O. 9764,
c/o P. M., San Francisco, Calif. ALR
Myers, Elmer A., '20.Board of Trade bldg., Superior, Wis. OALR
Myers, E. Lee, '26.....3903 Olive, St. Louis 8, Mo. ALR
Myers, John L., '20....Shukert bldg., Kansas City 6, Mo. OALR
Myers, Stanley A., '39, Lt. (M.C.), U.S.N.R.,
Address Unknown ALR
Myerson, M. C., '23.....136 E. 64th, New York 21, N. Y. ALR

N

②Nadeau, E. G., '15.....Belin bldg., Green Bay, Wis. **OALR**
②Naftzger, J. B., '17
6777 Hollywood blvd., Hollywood 28, Calif. **OALR**
③Nager, Prof. F., '25.Freiestrasse 20, Zurich, Switzerland **OALR**
Nail, James B., '36..1300 Eighth st., Wichita Falls, Texas **ALR**
Nairn, George Waverly, '41, Wyoming County
Community Hospital, Warsaw, N. Y. **ALR**
①Nance, Willis O., '03...6845 Euclid av., Chicago 49, Ill. **OALR**
Nash, C. Stewart, '29...277 Alexander, Rochester 7, N. Y. **ALR**
Nash, Herbert T., '37.....31 N. State, Chicago 2, Ill. **ALR**
Nebinger, Reid, '33.....16 W. Market, Danville, Pa. **ALR**
Neblett, Herbert Clarence, '40
401 North Tryon, Charlotte 2, N. C. **OP**
Neely, Paul T., '36...Medical Arts bldg., Portland 5, Ore. **ALR**
②Neeper, Edward R., '18
Exch. Nat'l Bk. bldg., Colorado Springs, Colo. **OP**
Neer, Edmonde DeW., '36.115 E. 61st, New York 21, N. Y. **ALR**
Neff, E. Eugene, '32.224 W. Washington, Madison 3, Wis. **OP**
Neher, Edwin M., '26
475 El Cam. Del Mar, Laguna Beach, Calif. **OP**
Neidlinger, William J., '42, Lt. (M.C.),
Finney Gen. Hosp., Thomasville, Ga. **ALR**
Neller, Walter I., '33....121 Wickham, Middletown, N. Y. **OP**
②Nelson, Charles F., '19
Schofield bldg., Cleveland 15, Ohio **OALR**
Nelson, Leo A., '30.....4105 Live Oak, Dallas 4, Texas **ALR**
②Nelson, Louis A., '18
Lowry Med. Arts bldg., St. Paul 2, Minn. **OP**
Nelson, Nathaniel F., '40.4910 15th av., Brooklyn 19, N.Y. **OP**
Nelson, Orville N., '36
1035 39th av. North, St. Petersburg 6, Fla. **OALR**
Nemours, Paul R., '29
1506 Hodiamont av., St. Louis 12, Mo. **ALR**
Nerbonne, Joseph J., '39.....231 Main, Brockton 1, Mass. **OP**
Nesbit, Mark E., '39
616 1st Nat'l Bank bldg., Madison 3, Wis. **OP**
Nesbit, Wellwood, '36.....1 S. Pinckney, Madison 3, Wis. **ALR**
Nethercut, G. W., '35...25 E. Washington, Chicago 2, Ill. **OP**
Nethercut, Ruth A., '38.450 Sutter, San Francisco 8, Calif. **ALR**
Neuberger, Julius Frederick, '42, Capt. (M.C.), U.S.N.,
U. S. Naval Hospital, Brooklyn, N. Y. **ALR**
Neumann, Wm. H., '28
Sheboygan Clinic, Sheboygan, Wis. **OALR**
Neuschatz, Gerald, '37
327 Central Park West, New York 25, N. Y. **ALR**
New, Gordon B., '27.....Mayo Clinic, Rochester, Minn. **L-PI S**
Newell, Frank W., '43, Lt. (M.C.),
A.P.O. 152, c/o Postmaster, New York, N. Y. **OP**
①Newhart, Horace, '05
Medical Arts bldg., Minneapolis 2, Minn. **ALR**
Newlands, W. A., '34....359 East Main, Mt. Kisco, N. Y. **ALR**
and 19 S. Broadway, Tarrytown, N. Y.
Newman, Erwin W., '38...1606 Capitol av., Cheyenne, Wyo. **OP**
Newton, F. H., '25....Medical Arts bldg., Dallas 1, Texas **OALR**
Newton, Morris H., '34...Burrell bldg., Little Falls, N. Y. **OP**
Newton, Ray E., '43..1010 Reynolds bldg., Jackson, Mich. **OP**
Nickle, Millen Alexander, '40....Box 305, Clearwater, Fla. **ALR**
Nippe, Leonard, '32.....316 Michigan, Toledo 2, Ohio **OALR**
②Nisselson, Max, '18
250 Riverside drive, New York 25, N. Y. **ALR**
Nodine, Edwin Rogers, '42
1110 Bell bldg., Montgomery 4, Ala. **ALR**
Noe, Carl A., '41, Major (M.C.), Army and Navy General
Hospital, Hot Springs, Ark. **OALR**
Noonan, W. J., '25.....4753 Broadway, Chicago 40, Ill. **OALR**
Nordstrom, Chester A., '37
Exchange Bank bldg., Franklin, Pa. **ALR**
Norris, Mary Alice, '43
404 Hume-Mansur bldg., Indianapolis 4, Ind. **OP**
Northcott, Thomas A., '26.40 E. 61st, New York 21, N. Y. **OP**
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Corps Air Station, Cherry Point, N. C. **ALR**
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1726 Eye st. N.W., Washington 6, D. C. **ALR**
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Nutting, Raymond J., '39..411 Thirtieth, Oakland 9, Calif. **OP**

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307 Sterling place, Brooklyn 17, N. Y. **ALR-PI S**
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②O'Connor, D. F., '18.....671 Broad, Newark 2, N. J. **OALR**
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O'Connor, Timothy Francis, '40
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Fort Hayes, Columbus 18, Ohio **OALR**
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Medicine S.A.A.A.B., Santa Ana, Calif. **ALR**
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Officer Procurement, 33 Pine, New York, N. Y. **ALR**
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2065 Grand Concourse, New York 53, N. Y. **ALR**
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Lowry Medical bldg., St. Paul 2, Minn. OP
①O'Reilly, W. F., '13.....44 S. Common, Lynn, Mass. OALR
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U. S. Naval Hospital, N.O.B., Norfolk 11, Va. ALR
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1350 Union, Schenectady 8, N. Y. OP
O'Toole, Francis Austin, '40..181 Chestnut, Clinton, Mass. ALR
Otrich, Grover C., '32....Commercial bldg., Belleville, Ill. ALR
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809 Hume-Mansur bldg., Indianapolis 4, Ind. ALR
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Box 1, Craigsville, N. Y. ALR
Owen, Walter E., '43...1242 Jefferson bldg., Peoria 2, Ill. ALR
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Col. (M.C.), 214 N. High, Hartford City, Ind. ALR
Owsley, Robert W., '41, Major (M.C.),
214 N. High, Hartford City, Ind. ALR

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General Hospital, Springfield, Mo. OALR
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Station Hospital, Lincoln Air Base, Lincoln 1, Nebr. ALR
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②Patterson, Ellen J., '17, (Retired)
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③Patterson, Major Gen. Robert U., '33, U. S. Army,
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②Patterson, W. E., '19
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Station Hospital, Army Air Field, Walnut Ridge, Ark. OP
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School of Aviation Medicine, Randolph Field, Tex. OP
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Simmons Nat'l bldg., Pine Bluff, Ark. ALR
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Pearson, Ovid A., '37, Major (M.C.), 0253096
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1930 Wilshire blvd., Los Angeles 5, Calif. ALR
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556 Purdue, University City, St. Louis County, Missouri ALR
Pember, Aubrey H., '26
500 W. Milwaukee, Janesville, Wis. OALR
Pendexter, R. Stevens, '26
Farragut Med. bldg., Washington 6, D. C. OP
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Station Hospital S.B.A.A.F., San Bernardino, Calif. ALR
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Perera, Charles A., '36...70 E. 66th, New York 21, N. Y. OP
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A.P.O. 186, c/o Postmaster, Los Angeles, Calif. OP
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40 S. 3rd st., Columbus 15, Ohio OP
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609 Lafayette Life bldg., Lafayette, Ind. ALR
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65 Pondfield Road, Bronxville, N. Y. ALR
Petti, George Hyacinth, '40.4 Berkeley av., Yonkers, N. Y. ALR
①Pfaffin, C. A., '03
Bankers Trust bldg., Indianapolis 4, Ind. OALR

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635 W. 165th, New York 32, N. Y. **OP**
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Station Hospital, Camp Livingston, La. **OP**
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Standish, Port of Embarkment, Boston, Mass. **ALR**
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16130 Parkland Drive, Shaker Heights 20, Ohio **OP**
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U. S. N. Academy, Annapolis, Md. **OP**
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Medical Arts bldg., Newport News, Va. **ALR**
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2909 Estes av., Chicago 45, Ill. **ALR**
Pollock, Frederic J., '38, Lt. (M.C.), U.S.N.R.,
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Lakes Naval Hospital, Great Lakes, Ill. **ALR**
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Mayo Clinic, Rochester, Minn. **OP**
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Physicians & Surgeons bldg., Minneapolis 2, Minn. **OALR**
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195 Hempstead av., Rockville Centre, L. I., N. Y. **OP**
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Professional bldg., Harrisonburg, Va. **ALR**
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Station Hospital, W.A.A.F., Waco, Texas **OP**
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Q

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Quinn, John L., '39.....401 Market, Steubenville, Ohio **ALR**
Quinn, Lester H., '37.....4105 Live Oak, Dallas 4, Texas **OP**
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R

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358 New York av., Brooklyn 13, N. Y. **ALR**

Rabinowitz, Paul, '38
Mountain Sanitarium, Hamilton, Can. **ALR**

Rafferty, George E., '38.....173 N. Common, Lynn, Mass. **ALR**

Rahmanop, Walter B., '39...1426 Elm, Manchester, N. H. **ALR**

Raines, Morris M., '43.....34 N. Ash, Ventura, Calif. **ALR**

②**Ralls, C. T.,** '16.....State Bank bldg., Winfield, Kan. **OALR**

Ralston, Frank L., '33...Sacajawea Annex, La Grande, Ore. **OALR**

②**Ralston, Wm. W.,** '18.1304 Walker av., Houston 2, Tex. **OP**

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Flight Lt. R.C.A.F., 320 Yale av., Winnipeg, Can. **OP**

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Medical Arts bldg., Oklahoma City 2, Okla. **OP**

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Ludlow and Heather Road, Upper Darby, Pa. **OP**

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①**Ravdin, M.,** '11.1666 Bayard Pk. Dr., Evansville 14, Ind. **OALR**

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384 Post, San Francisco 8, Calif. **ALR**

Rawlins, Grove A., '39.....206A West 3rd, Alton, Ill. **ALR**

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2835 4th av., Beaver Falls, Pa. **ALR**

Reed, James R., '37
201 N. El Molino av., Pasadena 4, Calif. **OP**

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Reeder, James E., Jr., '42, Capt. (M.C.),
523 Davidson bldg., Sioux City 13, Iowa **OP**

②**Reeder, J. E.,** '16...Davidson bldg., Sioux City 13, Iowa **OALR**

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Station Hospital, Randolph Field, Texas **OP**

Rees, Charles W., '38, Lt. Comdr. (M.C.),
4496 Trias, San Diego 3, Calif. **ALR**

Reese, Algernon B., '28...73 E. 71st, New York 21, N. Y. **OP**

Reese, Warren S., '21....1901 Walnut, Philadelphia 3, Pa. **OP**

Regan, James J., '32
520 Commonwealth av., Boston 15, Mass. **OP**

Regan, William F., '37.19 Bay State road, Boston 15, Mass. **ALR**

②**Reiche, O. C.,** '18.....Markle Bank bldg., Hazleton Pa. **OP**

Reid, H. W., '30...Union Central bldg., Cincinnati 2, Ohio **OP**

Reid, Thomas F., '40.....538 Central av., Dover, N. H. **ALR**

Reifsneider, J. Stanley, '38..Beacon bldg., Wichita 2, Kan. **ALR**

Reim, Hugo, '20.....Metropolitan bldg., St. Louis 3, Mo. **OP**

Reimers, R. S., '35....613 Eighth st., Fort Madison, Iowa **ALR**

Reineke, George F., '26...Fritsche bldg., New Ulm, Minn. **OALR**

Repass, R. E., '20....605 Lincoln road, Miami Beach, Fla. **ALR**

Reuling, F. H., '24.....Black bldg., Waterloo, Iowa **OALR**

Reuter, Carl H., '39
First Nat'l Bank bldg., Springfield, Ohio **OP**

Reynolds, Edna M., '37..227 Sixteenth st., Denver 2, Colo. **OP**

①**Reynolds, H. G.,** '09
Citizens Sav. Bank bldg., Paducah, Ky. **OALR**

Rhode, Homer J., '38.....220 N. 6th, Reading, Pa. **OP**

Rhode, Solon L., '28.....238 N. 6th, Reading, Pa. **OALR**

Rhodes, David H., '37.....Clark bldg., Pittsburgh 22, Pa. **OP**

Ricchiuti, A. George, '41, Capt. (M.C.), Sta. Hosp.,
Avon Park Bomb Range, Avon Park, Fla. **ALR**

Rice, Carl E., '38....6500 Maple av., Chevy Chase 15, Md. **OP**

Rice, John E., '24.....390 Main, Worcester 8, Mass. **OALR**

Richards, Lyman G., '24, Lt. Comdr. (M.C.), U.S.N.R.,
15 Whiting rd., Wellesley Hills, Mass. **ALR**

Richardson, George A., '38
1532 Overington, Philadelphia 24, Pa. **ALR**

Richardson, George A., '42, In Service, Address Unknown **ALR**

Richardson, J. J., '29
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Robb, Mayo, '29.....Professional bldg., Phoenix, Ariz. **ALR**

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111 S. Huron av., Pierre, S. D. **ALR**

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Rudolphy, J. B., '29.....230 S. 21st, Philadelphia 3, Pa. **OP**
Ruedemann, A. D., '29
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Station Hospital, Camp Gordon, Augusta, Ga. **ALR**

Shearer, Harry A., '39.....405 E. Grand av., Beloit, Wis. **ALR**

②**Shellman, John L.,** '17
2731 Washington av., Santa Monica, Calif. **OP**

②**Shemeley, William G., Jr.,** '19
7 Haddan av., Camden, N. J. **OALR**

Shepard, Emerson P., '37..72 W. Second, Chillicothe, Ohio **ALR**

Shepard, Warren Brundage, '41
Jenkins Arcade, Pittsburgh 22, Pa. **ALR**

Shepherd, Elmer Roberts, '41
1746 K st. N.W., Washington 6, D. C. **ALR**

Sheppard, Ernest A. W., '36
927 17th st. N. W., Washington 6, D. C. **OP**

Sheppard, Louis B., '37, Major (M.C.),
Seymour Johnson Field, Goldsboro, N. C. **OP**

Sher, Maurice A., '38
5022 Fifteenth av., Brooklyn 19, N. Y. **ALR**

①**Sherman, A. Edward,** '13, Lt. Col. (M.C.), (retired),
Route 1, Box 1401, La Mesa, Calif. **OALR**

Sherman, Arthur E., '39
48 Headley Place, Maplewood, N. J. **OP-PI S**

Sherman, A. Russell, '33.....671 Broad, Newark 2, N. J. **OP**

①**Sherman, Elbert S.,** '12.....671 Broad, Newark 2, N. J. **OP**

Sherman, R. Newton, '34
Phoenix bldg., Bay City, Mich. **OALR**

②**Shields, J. M.,** '15...Metropolitan bldg., Denver 2, Colo. **OP**

Shilling, Raymond L., '42, Capt. (M.C.),
Station Hospital, Army Air Base, Reno, Nevada **ALR**

Shipley, J. L., '33, Capt. (M.C.),
Pre-Flight School, Iowa City, Iowa **OALR**

Shipman, James S., '35, 514 Cooper, Camden, N. J.
and 255 S. 17th, Philadelphia 3, Pa. **OP**

Shires, Howard H., '20
Guardian bldg., Cleveland 14, Ohio **OP**

Shlossberg, Frank Ronald, '41
116 Emerson st., Haverhill, Mass. **ALR**

Shmukler, B. Cecelia, '41
1930 Wilshire blvd., Los Angeles 5, Calif. **OP**

Shoemaker, Ferdinand, '20
3232 Harrison, Kansas City 3, Mo. **OALR**

②**Shoemaker, S. A.,** '17....613 S. Hyer st., Orlando, Fla. **OALR**

Shope, Edward Pierce L., '32..511 Cooper, Camden, N. J. **OP**

Shorr, Abraham, '40
225 Eastern Parkway, Brooklyn 16, N. Y. **ALR**

①**Shreve, Owen M.,** '05.....162 W. 8th, Erie, Pa. **OP**

Shumaker, Phil W., '39
9730 Wilshire blvd., Beverly Hills, Calif. **ALR**

②**Shuman, G. H.,** '17....351 Fifth av., Pittsburgh 22, Pa. **OP**

①③**Shurly, Burt R.,** '03
62 Adams av. W., Detroit 26, Mich. **ALR**

Shurtleff, Raymond S., '41.....Box 757, Lynch, Ky. **ALR**

Shuster, B. H., '29.....1824 Pine, Philadelphia 3, Pa. **ALR**

Sidwell, C. E., '29.....608 Fourth av., Longmont, Colo. **ALR**

Siegel, Francis X., '22
Union Central Life bldg., Cincinnati 2, Ohio **OALR**

Siewert, Otto Lucht, '42, Capt. (M.C.), 0-468093,
A.P.O. 645, c/o P. M., New York, N. Y. OP
Siberman, Meyer, '38.266 Brooklyn av., Brooklyn 13, N. Y. ALR
②Sibernagel, C. E., '18..247 E. State, Columbus 15, Ohio OALR
Silcox, Louis Edward, '40, Lt. (M.C.), U.S.N.R.,
U. S. Naval Hosp., N.O.B., Norfolk, Va. ALR
Silva, Rafael, '32.Av. Insurgentes 195, Mexico City, Mexico OP
Silverberg, Joseph S., '39
755 Ocean av., Brooklyn 26, N. Y. ALR
Silverberg, Samuel Joshua, '40
201 Park st., New Haven 11, Conn. ALR
Simkins, James J., '37..1737 Chestnut, Philadelphia 3, Pa. ALR
Simmonds, Noel T., '38.....1404 Murray, Alexandria, La. OP
Simmons, J. Thomas, '43.....316 W. 10th st., Erie, Pa. OP
Simon, Emanuel, '32, Lt. Comdr. (M.C.),
Navy 115, F. P. O., New York, N. Y. ALR
Simons, Leander T., '42, Lt. Col. (M.C.),
Station Hospital, Ft. Leonard Wood, Mo. OP
Simons, Max, '41, Capt. (M.C.),
P.O.W. Camp, Concordia, Kan. ALR
Simons, N. S., '30....MacCornack Clinic, Whitehall, Wis. ALR
Simonton, Kinsey M., '41, Major (M.C.), 0-305888,
APO 322, c/o Postmaster, San Francisco, Calif. ALR
Simpson, G. Victor, '31, Lt. Col. (M.C.),
Nashville Army Air Center, Nashville 4, Tenn. OP
Simpson, H. Lee, '21
David Whitney bldg., Detroit 26, Mich. ALR
Simpson, John R., '27..Med. Arts bldg., Pittsburgh 13, Pa. ALR
Simpson, M. B., '30..Professional bldg., Kansas City 6, Mo. ALR
Simpson, Robert K., '39, Col. (M.C.),
455 Lake av., St. Louis 8, Mo. OP
①Simpson, W. Likely, '11
Physicians and Surgeons bldg., Memphis 3, Tenn. OALR
Sims, J. Lawrence, '42, Capt. (M.C.),
Station Hospital, Selfridge Field, Mich. ALR
Sinamark, Andrew, '28.....Power bldg., Hibbing, Minn. OALR
Singleton, John D., '37....1719 Pacific av., Dallas 1, Texas ALR
Sink, Vergil Rex, '41.852 W. 5th, Winston-Salem 5, N. C. ALR
Sitchevska, Olga L., '32..30 Fifth av., New York 11, N. Y. OP
Sitney, Julian J., '43, Lt. (M.C.), Station Hospital
Charlestown Army Air Base, Charlestown, S. C. OP
Skillern, S. R., Jr., '25.....1734 Pine, Philadelphia 3, Pa. ALR
Skirball, Joseph J., '27
353 Commonwealth av., Boston 15,, Mass. OP
Skow, John D., '39...2001 Collingwood av., Toledo 2, Ohio OP
Slack, Walter K., '34, Comdr. (M.C.),
U. S. Naval Hospital, Seattle 4, Wash. ALR
②Sloan, Henry L., '19.....106 W. 7th, Charlotte 2, N.C. OP
Sloane, Albert E., '38..416 Marlborough, Boston 15, Mass. OP
Sloane, H. O., '30.....1717 Pine, Philadelphia 3, Pa. OP
Sly, John L., '33.....382 Springfield av., Summit, N. J. ALR
Small, Mary Louise, '38.....18 W. Read, Baltimore 1, Md. OP
Smart, Robert Edwin, '42
Medical Arts bldg., Ottawa, Ontario, Can. OP
Smiley, William A., '38, Major (M.C.),
66th Field Hospital, Camp Ellis, Ill. ALR
Smit, William M., '28
5285 Washington blvd., St. Louis 8, Mo. ALR
Smith, Alma C., '36, Capt. (M.C.),
Navy 135, Fleet P. O., San Francisco, Calif. OALR
Smith, Arthur E., '20,
Medical Arts bldg., Minneapolis 2, Minn. OALR
Smith, Austin T., '39, Comdr. (M.C.),
Norfolk Naval Hosp., Portsmouth, Va. ALR
Smith, Carlie Souter, '27...Holland bldg., Springfield, Mo. OALR
Smith, Carroll L., '29
Paulsen Med. & Dental bldg., Spokane 8, Wash. OALR
Smith, Clarence H., '37..140 E. 54th, New York 22, N. Y. ALR
②Smith, D. S., '18....State Bank bldg., La Crosse, Wis. OALR
①Smith, Dorland, '10..881 Lafayette, Bridgeport 4, Conn. OP A
①Smith, E. Terry, '12..P. O. Box 42, West Hartford, Conn. OP
Smith, Ernest Gerard, '41.339 N. Duke, Lancaster, Pa. OP
Smith, Eugene C., '36
Union Industrial bldg., Flint 3, Mich. OALR
Smith, Ferris, '33
Blodgett Med. bldg., Grand Rapids 6, Mich. ALR-PI S
Smith, Franklin C., '39
Charlotte E.E.&T. Hosp., Charlotte 2, N. C. OP
①Smith, George B., '14.....Harbin Hosp., Rome, Ga. OALR
Smith, George H., '38, Maj. (M.C.),
A.P.O. 720, c/o Postmaster, San Francisco, Calif. ALR
Smith, H. A., '25..226 N. Greenleaf av., Whittier, Calif. OALR
Smith, H. Brooks, '40.....303 S. Main, Bluffton, Ind. OP
Smith, Harold D., '39, Capt. (MC)
School of Aviation Medicine, Randolph Field, Texas ALR
Smith, Henry B., '37.131 Fulton av., Hempstead, N. Y. ALR
Smith, Henry C., '38, Lt. Col.
(MC), McCloskey General Hospital, Temple, Texas OP
Smith, Herschel S., '42, Major (MC),
Med. Det. 411th Inf., A.P.O. 470, Camp Howze, Texas OP
Smith, Hiram J., '38..30 N. Michigan av., Chicago 2, Ill. OP
Smith, I. Ruebert, '39....116 College st., Toronto, Can. ALR
Smith, J. Allen, '39.....Doctors bldg., Macon, Ga. ALR
Smith, James M., '31..123 E. 53rd, New York 22, N. Y. ALR
Smith, James W., '37..1016 Fifth av., New York 28, N. Y. OP
Smith, Jay D., '38 Maj. (MC), Box 6,
Station Hospital No. 2, Fort Jackson, S. C. ALR
Smith, Josiah E., '39..60 Meeting st., Charleston 1, S. C. OALR
Smith, Leon, '39.....467 Linwood av., Buffalo 9, N. Y. ALR
Smith, Leslie M., '43, Lt. Comdr. (MC),
U. S. Marine Hospital, Baltimore 11, Md. OP
Smith, Morley T., '36..421 Huguenot, New Rochelle, N. Y. ALR
Smith, Norman N., '43
291 Whitney av., New Haven 11, Conn. ALR
Smith, R. N., '29....Medical Arts bldg., Tulsa 3, Okla. ALR
Smith, R. O., '37.....73 Third av. N. W., Pulaski, Va. ALR
Smith, Robert Arvil, '41, Lt. Col. (M.C.), Box 6063,
Fitzsimmons General Hospital, Denver, Colo. ALR
Smith, Rodger Browning, '42
224 W. Washington av., Madison 3, Wis. ALR
Smith, Thomas T., '43, Lt. (M.C.),
Norfolk Naval Hospital, Portsmouth, Virginia ALR
①Smith, Victor C., '12
American Bank bldg., New Orleans 12, La. OP
Smith, W. Hamilton, '36
314 Wareham bldg., Hagerstown, Md. ALR
Smith, W. Jewell, '39, Lt. Comdr. (M.C.),
Lion 6, APBD., U. S. Navy, San Bruno, Calif. ALR
Smith, William L., '30
Medical Arts bldg., Salt Lake City 1, Utah OP
Smith, Windsor R., '33..67 Front, Binghampton, N. Y. ALR
Smithies, Harold R., '37..2241 Central av., Alameda, Calif. ALR
Smoot, Merrill C., '39, Lt. Comdr. (M.C.), USNR,
Camp LeJuene, New River, N. C. ALR
Snapp, Carl F., '28
Medical Arts bldg., Grand Rapids 2, Mich. ALR
Sneed, Carl M., '20.....Exchange bldg., Columbia, Mo. OALR
Snell, Albert C., '25..53 S. Fitzhugh, Rochester 4, N. Y. OP
Snell, George C., '33, Medical Arts bldg., Toronto, Can. ALR
Snelling, P. W., '31.....179 Allyn, Hartford 3, Conn. ALR
Snitman, Maurice F., '42
55 E. Washington st., Chicago 2, Ill. ALR
Snow, Harold L., '40.....639 W. 9th, San Pedro, Calif. OP

Snydacker, Daniel, '38, Major (M.C.),
Dibble General Hospital, Menlo Park, Calif. OP
Snyder, Eugene Ralph, '40, Lt. Comdr. (M.C.),
Marine Air Station, M.A.G. 34, Cherry Point, N. C. ALR
Snyder, George Gordon, '40
Mermont Apartments, Bryn Mawr, Pa. ALR
Snyder, Jerome, '38, Lt. (M.C.),
103rd Evacuation Hospital, Fort Lewis, Wash. ALR
Snyder, William S., Jr., '36...324 W. Main, Frankfort, Ky. ALR
Soboroff, Simon H., '40
1608 Milwaukee av., Chicago 47, Ill. ALR
Sole, Adrian, '40....174 Highland av., Somerville, Mass. ALR
Solomon, Henry D., '39
Power & Light bldg., St. Petersburg 5, Fla. ALR
Somberg, J. S., '23...406 Bulkley bldg., Cleveland 15, Ohio OP
Soper, Gail R., '33.....636 Church, Evanston, Ill. OP
Sowers, Alva B., '37
6 N. Michigan av., Sec. 2, Chicago 2, Ill. OP
Spaeth, Edmund B., '28
1930 Chestnut, Philadelphia 3, Pa. OP-PI S
Spake, LaVerne B., '20
Brotherhood bldg., Kansas City 11, Kans. ALR
Spalding, Lansford M., '39
185 Fourteenth st., Astoria, Ore. ALR
Sparber, Herman, '35
4701 Fifteenth av., Brooklyn 19, N. Y. OP
Sparer, William S., '39....50 E. 10th, New York 3, N. Y. ALR
Sparks, Alan Leo, '40
Maj. (M.C.), 4310 Central av., Indianapolis 5, Ind. ALR
Spearman, Maurice P. S., '42,
1315 First Nat'l Bank bldg., El Paso, Texas ALR
Speas, William Paul, '41
Reynolds bldg., Winston-Salem 3, N. C. OP
①Spencer, Frank R., '04..Physicians bldg., Boulder, Colo. OALR
Spencer, George A., '28
1025 Ninth st., Sacramento 14, Calif. OALR
Spencer, S. C., '29.....Professional bldg., Waco, Texas OALR
②Spencer, W. H., '18
1830 S. Rittenhouse sq., Philadelphia 3, Pa. ALR
Spencer, Walter Carlton Edward, '40
Medical Arts bldg., Houston 2, Tex. OP
②Spengler, John A., '16.....423 S. Main, Geneva, N. Y. OP
Sperry, F. N., '36..107 Whitney av., New Haven 10, Conn. ALR
Spiegelglass, A. B., '39.....417 Main, Hackensack, N. J. ALR
Spiesman, I. G., '29, 1908 St. Charles road, Maywood, Ill. ALR
②Spitze, E. C., '16....Murphy bldg., East St. Louis, Ill. OP
Sporn, Hyman, '38, Maj. (M.C.), Station Hospital,
Camp Wheeler, Macon, Ga. ALR
Springer, James Earl, '41
Peoples Bank bldg., Akron 8, Ohio ALR
Sproule, Ralph P., '28
208 E. Wisconsin, Milwaukee 2, Wis. OALR
Sputh, Carl B., '40
224 N. Meridian st., No. 301, Indianapolis 4, Ind. ALR
Stack, James Edward, '41
8838 Woodhaven blvd., Woodhaven 21, N. Y. ALR
①Stanbery, Henry, '10..Doctors bldg., Cincinnati 2, Ohio OP
Stanford, Charles E., '39
Medical Arts bldg., Minneapolis 2, Minn. OP
②Stanford, J. B., '16..899 Madison av., Memphis 3, Tenn. OP
Stanley, C. R., '30....508 Tenth st., Worthington, Minn. ALR
Stanton, Harmon L., '28, Hulman bldg., Evansville 18, Ind. OALR
Stark, Jesse B., '26.....46 Park av., New York 16, N. Y. OP
Stark, W. Berkeley, '27..Medical Arts bldg., Toronto 5, Can. ALR
Statti, Louis Wm., '37..Jenkins Arcade, Pittsburgh 22, Pa. OP
Stauffer, F. Leaver, '34
52 East South Temple st., Salt Lake City 1, Utah ALR
Stauffer, Harry B., '43
234 Madison, Jefferson City, Missouri ALR
Steele, Willard, '28.....721 Broad, Chattanooga 2, Tenn. OALR
Steffensen, W. H., '37, Capt. (M.C.), Station Hospital,
Sheppard Field, Wichita Falls, Tex. ALR-PI S
Steffenson, Olaf, '38....25 E. Washington, Chicago 2, Ill. ALR
①Steim, J. M., '14
First Nat'l Bank bldg., New Kensington, Pa. OP
Steinbach, Louis, '37
1882 Grand Concourse, New York 57, N. Y. OP
Steinberg, Martin R., '41, Maj. (M.C.),
Patterson Field, Fairfield, Ohio ALR
Steinburgler, Wm. F. C., '35
515 Ocean av., Brooklyn 26, N. Y. OP
Steinfield, A. L., '22....125 Fifteenth st., Toledo 2, Ohio OALR
Stephan, Lewis B., '41, Capt. (M.C.),
438 Brooks st., Missoula, Montana OP
Stern, Milton J., '35
First Nat'l Bank bldg., Lexington 3, Ky. OP
Stern, Wilhelm E., '41
1099 Bushwick av., Brooklyn 21, N. Y. ALR
Stevens, Ralph W., '38, Capt. (M.C.),
Station Hospital, Stout Field, Indianapolis 6, Ind. OP
②Stevenson, Walter, '15.....W. C. U. bldg., Quincy, Ill. OALR
Stevenson, Walter D. Jr., '43, Major (M.C.),
McCloskey General Hospital, Temple, Texas ALR
Stevenson, William W., '31, Kresge bldg., Flint 3, Mich. OALR
Stewart, Charles E., '43, Capt. (M.C.),
Percy Jones General Hospital, Battle Creek, Mich. ALR
Stewart, James K., '37, Comdr. (M.C.),
U. S. Naval Hosp., Bainbridge, Md. ALR
Stewart, James V., '42, Capt. (M.C.),
Columbus Army Air Base, Columbus, Ohio ALR
①Stewart, T. M., '20
Union Central bldg., Cincinnati 2, Ohio OP
Stewart, Walter E., '20, Tribune bldg., Terre Haute, Ind. OALR
①Stieren, Edward, '04, Union Trust bldg.,
Pittsburgh 19, Pa. OP
①Stiles, Charles M., '08
334 W. Wiona av., Philadelphia 44, Pa. OP
①Stillson, H., '03.....Seabord bldg., Seattle 1, Wash. OALR
②Stimson, George W., '19.580 Perkins drive, Warren, Ohio OALR
Stine, George H., '32..Burns bldg., Colorado Springs, Colo. OP
Stinson, Wm. D., '28..899 Madison av., Memphis 3, Tenn. ALR
Stitt, H. L., '27.....Doctors bldg., Cincinnati 2, Ohio ALR
Stockwell, Eunice LeBaron, '42
2018 Pine st., Philadelphia 3, Pa. OP
Stoffregen, Charles Henry, '41
10515 Carnegie av., Cleveland 6, Ohio ALR
Stokes, Wm. H., '26.Medical Arts bldg., Omaha 2, Nebr. OP
Stoll, K. Ludwig, '29.....19 W. 7th, Cincinnati 2, Ohio OP
Stone, Harry B., '30..Medical Arts bldg., Roanoke 11, Va. ALR
Stone, Harry B., Jr., '39
Lt. (M.C.), U. S. Naval Hosp., Coco Solo, Canal Zone ALR
Stone, Leslie O., '35, Capt. (M.C.),
U. S. Naval Disp., Submarine Base, New London, Conn. ALR
Stonecypher, D. D., '30
102½ S. 9th st., Nebraska City, Nebraska ALR
Stoner, Cyrus Hubert, '42, Maj. (M.C.),
Box 523, Chanute Field, Illinois OP
Stoner, Donald D., '43...156 W. Pomfret st., Carlisle, Pa. ALR
②Stotter, Arthur L., '16
1148 Euclid av., Cleveland 15, Ohio OALR
Stough, John T., '39..Medical Arts bldg., Houston 2, Texas OP

Stoutenborough, Wm. A., '27
21 E. State, Columbus 15, Ohio OALR
Stovin, Joseph S., '36
151 Central Park West, New York 23, N. Y. ALR
Straatsma, Clarence R., '32, Lt. Comdr. (M.C.),
66 E. 79th st., New York 21, N. Y. ALR-PI S
Straus, Gerhard David, '41, Capt. (M.C.),
Box 119, Chanute Field, Rantoul, Ill. ALR
Strause, Harold L., '43
826 N. 5th st., Reading, Pennsylvania ALR
Strauss, Arthur, '43
130 Pavilion av., Long Branch, New Jersey ALR
Strauss, J. F., '25....104 S. Michigan av., Chicago 3, Ill. ALR
Street, Alvin Murray, '41.745 5th av., New York 22, N. Y. ALR
Streit, August J., '31.....724 Polk, Amarillo, Texas ALR
Strickler, Alfred D., '43.....26 N. 9th st., Lebanon, Pa. OP
Strickler, Joseph G., '37.200 W. 57th, New York 19, N. Y. ALR
Strikol, Albert J., '36
621 Delaware av., Wilmington 9, Delaware ALR
Strong, James Craig, Jr., '42, Capt. (M.C.),
Hq. Reno A.A.B., Reno, Nev. OP
Stroud, Sanders Key, '40
Capt. (M.C.), Borden General Hosp., Chickasha, Okla. OP
Strout, G. Elmer, '21
1009 Nicollet av., Minneapolis 2, Minn. OALR
Struble, Gilbert Carl, '42, Lt. Col. (M.C.), Billings
General Hospital, Fort Benjamin Harrison, Ind. OP
Stuard, Charles G., '43, Major (M.C.),
Station Hospital, Jefferson Barracks, Mo. OP
①Stueber, F. G., '05.....209 W. North, Lima, Ohio OALR
②Stueber, Paul J., '17.....209 W. North, Lima, Ohio OALR
②Sturges, Leigh F., '18..121 E. 60th, New York 22, N. Y. ALR
Stutsman, Albert C., '37
539 N. Grand blvd., St. Louis 3, Mo. ALR
Sudranski, Herbert F., '40
522 Hume Mansur bldg., Indianapolis 4, Ind. OP
Suehs, Oliver W., '41, Capt. (M.C.), 0-404556,
A.P.O. 511, c/o Postmaster, New York, N. Y. ALR
Sugar, H. Saul, '41, Major (M.C.),
Barnes General Hospital, Vancouver, Wash. OP
Sullivan, Charles T., '37..277 Alexander, Rochester 7, N. Y. OP
Sullivan, Clifford Patrick, '40, Capt. (M.C.),
304 W. 63rd st., Chicago 21, Ill. OP
Sullivan, Joseph A., '33, Wing Commander (RCAF),
Medical Arts bldg., Toronto 5, Ontario, Can. ALR
Sullivan, Robert E., '32...Doctors bldg., Nashville 3, Tenn. OP
Sullivan, Timothy, '42, Capt. (M.C.), 0-494925,
APO 929, c/o Postmaster, San Francisco, Cal. ALR
Sulzman, Frank M., '22....1831 Fifth av., Troy, N. Y. OALR
Sulzman, John H., '39, Lt. (M.C.), U.S.N.R.,
1831 Fifth av., Troy, N. Y. ALR
Summers, Wm. S. '27
David Whitney bldg., Detroit 26, Mich. OALR
Sunde, Einar A., '38.....627 Second, Brooklyn 15, N. Y. ALR
Sutherland, J. M., '29....Fisher bldg., Detroit 2, Mich. ALR
Sutherland, William S., '40
1930 Chestnut st., Philadelphia 3, Pa. OP
Swab, Charles M., '28, Medical Arts bldg., Omaha 2, Nebr. OP
Swaim, Charles J., '39
1330 W. Rockland, Philadelphia 41, Pa. ALR
Swan, John R., '39
915 Hume Mansur bldg., Indianapolis 4, Ind. ALR
Swan, Kenneth C., '43, University Hosp., Iowa City, Ia. OP
Swann, Cecil C., '32....Flat Iron bldg., Ashville, N. C. ALR
Swanson, Clifford A., '38, Comdr. (M.C.),
c/o Fleet Postoffice, New York, N. Y. ALR
②Swetman, C. R. K., '15
219 S. Mt. Vernon, Prescott, Ariz. ALR
Swick, David A., '37....30 W. 70th, New York 23, N. Y. OP
Swift, Donald E., '39, Capt. (M.C.),
O'Reilly General Hospital, Springfield, Mo. OP
Swigert, John L., '37, Maj. (M.C.),
Station Hospital, Fort Douglas, Utah OP
Swing, Frank U., '35...Doctors bldg., Cincinnati 2, Ohio ALR
Sykes, Clarence S., '39..2201 Market, Galveston, Texas OP
Sykes, Edwin Meredith, '42
Nix Professional bldg., San Antonio 5, Tex. ALR
Syracuse, Victor R., '41
29 Washington sq. W., New York 11, N. Y. OP

T

Tabor, Gilbert S., '37....11 Park Row, Millerton, N. Y. ALR
②Tangeman, H. F., '19
Union Central bldg., Cincinnati 2, Ohio ALR
Tangen, George M., '43
University of Minnesota, Minneapolis 14, Minn. ALR
Tanner, James W., '32.131 S. Barstow, Eau Claire, Wis. ALR
Taquino, George J., '20..Canal bldg., New Orleans 12, La. ALR
①Tarum, William, '10..104 W. Madison, Baltimore 1, Md. OP
Tassman, Isaac S., '29
Medical Arts bldg., Philadelphia 3, Pa. OP
Taylor, E. Merle, '31.Medical Arts bldg., Portland 5, Ore. OP
Taylor, H. Marshall, '35
111 W. Adams, Jacksonville 2, Fla. ALR
Taylor, Joseph W., '34.....706 Franklin, Tampa 2, Fla. OALR
②Taylor, M. C., '17.....May bldg., Pittsburgh 22, Pa. OP
Taylor, Shahane R., '39, Jefferson bldg., Greensboro, N. C. ALR
Teall, Raymond E. '28
Professional bldg., Kansas City 6, Mo. ALR
Tebbutt, H. K., '30.....240 State, Albany 6, N. Y. ALR
Teed, R. Wallace, '36, Lt. Comdr. (M.C.),
410 Highland road, Ann Arbor, Mich. ALR
Tegelberg, Julius J., '39, Lt. Comdr. (M.C.)
12 Monterey road, Worcester 6, Mass. ALR
Tegge, Charles W., '37
1801 Eye st. N. W., Washington 6, D. C.,ALR
Terrell, Vance, '42, Stephenville Hospital, Stephenville, Tex. ALR
Terry, Theodore L., '32
140 Marlborough, Boston 16, Mass. OP
Thacker, Edgar A., '43
602 W. University av., Urbana, Illinois ALR
Thaler, Nathan, '40
570 Eastern Parkway, Brooklyn 25, N. Y. ALR
Theis, Franklyn B., '39....209 S. Broadway, Nyack, N. Y. ALR
Thel, Henry C., '38.....387 Franklin, Aliquippa, Pa. ALR
Theobald, Georgiana Dvorak, '29
715 Lake, Oak Park, Ill. OP
Theobald, John J., '29.....715 Lake, Oak Park, Ill. ALR
Theobald, W. H., '30..307 N. Michigan av., Chicago 1, Ill. ALR
Theodore, Frederick H., '40, Capt. (M.C.),
1210 Lenox av., Miami Beach 39, Fla. OP
①Thigpen, C. A., '12....401 S. Court, Montgomery 5, Ala. OALR
Thoburn, Thomas W., '39
1020 Huron road, Cleveland 15, Ohio ALR
Thomas, Charles I., '43
10515 Carnegie av., Cleveland 6, Ohio OP
Thomas, Edward R., '37.....60 Wyoming, Dayton 9, Ohio OP
②Thomas, Francis W., '16 327 E. State, Columbus 15, Ohio OALR
Thomas, Harry Vernon, '40
Empire Bank bldg., Clarksburg, W. Va. OP
Thomas, James Alanson, '41
Southern Michigan Bank bldg., Coldwater, Mich. ALR
Thomas, James W., '28.....Sharp bldg., Lincoln 8, Nebr. OALR
Thomas, W. Maxwell, '35, Lt. Comdr. (M.C.),
437 W. Houston, Sherman, Texas OP

①Thomason, H. E., '12
Professional bldg., Kansas City 6, Mo. ALR
①Thompson, E. H., '07....19 W. 7th, Cincinnati 2, Ohio OALR
①Thompson, G. H., '09....74 North st., Pittsfield, Mass. OALR
Thompson, Howard E., '28.....1100 Main, Dubuque, Iowa OALR
Thompson, James Parvin, '42, Capt. (M.C.),
C.A.A.B., Station Hospital, Columbus, Miss. OP
Thompson, Lester Emmit, '42
Woolworth bldg., Salida, Colo. ALR
①Thompson, Peter H., '14
308 Commonwealth av., Boston 15, Mass. OP
Thompson, William R., '43, Lt. Col. (M.C.),
1550 Service Unit, Station Hosp., Fort Knox, Ky. ALR
①Thomsen-von Colditz, G., '06
Dun Huntin, Merritt Island, Fla. OALR
Thorburn, O. L., '39, Major (M.C.),
Army Air Base, Station Hospital, Alamogordo, N. M. ALR
Thornburgh, Robert Grant, '42
Professional bldg., Long Beach 2, Calif. OP
Thorne, Fred Sanborn, '39
270 Commonwealth av., Boston 16, Mass. OP
Thorpe, Harvey E., '31..Jenkins Arcade, Pittsburgh 22, Pa. OP
Thorson, John A., '36.....Roshek bldg., Dubuque, Iowa OP
Thygeson, Phillips, '34, Major (M.C.),
971 Eldorado, Clearwater Beach, Fla. OP
Tickle, Thomas G., '36.30 E. 40th st., New York 16, N. Y. ALR
Tiedke, Gunther E., '39, Lt. Comdr. (M.C.),
602 Dream's Landing, W. Annapolis, Md. OP
Tilderquist, D. L., '25...Med. Arts bldg., Duluth 2, Minn. OALR
Tillotson, R. S., '30
Medical-Dental bldg., Sacramento 14, Calif. ALR
①Timberman, Andrew, '03
91 Hamilton Park, Columbus 3, Ohio OALR
Tindal, John M., '33.....5517 W. Fulton, Chicago 44, Ill. OALR
①Tingley, Louisa Paine, '13
416 Marlborough, Boston 15, Mass. OP
Tinkess, Donald E., '43, Major (M.C.), Station Hospital
Box 42, Fort Jackson, S. C. OP
Tippin, Ernest E., '34
1st Nat'l Bank bldg., Wichita 2, Kan. ALR
①Tivnen, Richard J., '08
30 N. Michigan av., Chicago 2, Ill. OALR
②Tobey, George L., Jr., '17
403 Commonwealth av., Boston 15, Mass. ALR
Todd, Grover C., '22....Jenkins Arcade, Pittsburgh 22, Pa. ALR
Tous, Leopold T., '37.....967 Elm, Manchester, N. H. ALR
Tolan, John F., '38.....1115 Terry, Seattle 1, Wash. ALR
Tolan, Thomas L., '33.....Wells bldg., Milwaukee 2, Wis. ALR
Tolman, Henry, '39.....136 Federal, Salem, Mass. ALR
Tomassene, R. A., '23.....1144 Market, Wheeling, W. Va. OALR
Tomb, E. H., '30, Major (M.C.),
46 Lexington, Framingham, Mass. ALR
Tomlinson, John Wesley, '42, Lt. Comdr. (M.C.),
900 Chester Pike, Sharon Hill, Pa. OP
Tomlinson, Roland D., '39, 505 E. Broad st.,
Westfield, N. J. ALR
①Toms, S. W. S., '09....120 S. Broadway, Nyack, N. Y. OALR
②Tooker, C. W., '17....Carleton bldg., St. Louis 1, Mo. OP
Toot, J. F., '30.....First Nat'l Bank bldg., Canton 2, Ohio OP
Town, Arno E., '36, Lt. Comdr. (M.C.),
U. S. Naval Hospital, Bethesda, Md. OP
Townes, C. Dwight, '32, Lt. Col. (M.C.), Station Hospital,
S.A.A.C.C., San Antonio, Tex. OP
Townsend, John F., '23.85 Wentworth, Charleston 6, S. C. OALR
Towson, Charles E., '32
Germantown Prof. bldg., Philadelphia 44, Pa. ALR
Tracewell, George Logan, '41
Commerce bldg., Okmulgee, Okla. ALR
Tracht, Robert R., '41.....Lowry bldg., St. Paul 2, Minn. OP
Trainor, George M., '42, Capt. (M.C.),
A.P.O. 518, c/o Postmaster, New York, N. Y. ALR
Traugh, George H., '33
309 Cleveland av., Fairmont, W. Va. ALR
Tremble, George E., '32
1390 Sherbrooke st. W., Montreal, Can. ALR
Treskow, Frank G., '41...411 E. Mason, Milwaukee 2, Wis. ALR
Trexler, Clarence W., '37, Lt. Comdr. (M.C.),
50 Dowsett av., Honolulu, Hawaii ALR
Trible, G. B., '20
1150 Connecticut av. N. W., Washington 6, D. C. OALR
Trimble, Clarence S., '20....Gazette bldg., Emporia, Kan. OALR
Tripp, Leroy R., '20....Security bldg., Sioux City 8, Iowa OALR
Troncoso, Manuel U., '39
500 West End av., New York 24, N. Y. OP
Trott, Leslie De Nyse, '42
143 North Brand, Glendale 3, Calif. ALR
Trotter, Homer A., '32...40 W. North st., Buffalo 2, N. Y. ALR
Troup, Wilson J., '38, Lt. Comdr. (M.C.),
U. S. Naval Hospital, Bethesda, Md. ALR
Troutman, Erwin W., '40
21 East State st., Columbus 15, Ohio ALR
Trowbridge, Bernard C., '41, Lt. (M.C.),
Scott Field, Belleville, Ill. ALR
Trowbridge, Dwight H., '39
T. W. Patterson bldg., Fresno 1, Calif. OP
Trueman, Robert Heiserman, '42, Major (M.C.),
Stark General Hospital, Charleston, S. C. OP
Trull, Alfred C., '38
3 Washington Square, Haverhill, Mass. OP
Trumbo, James K., '37.....526 Third st., Wausau, Wis. ALR
Tucker, Frank Landale, Jr., '42
464 9th st., Brooklyn 15, N. Y. ALR
Tucker, Gabriel, '23.....250 S. 18th, Philadelphia 3, Pa. LB
①Tuckerman, W. H., '08
1020 Prospect av., Cleveland 15, Ohio OALR
Turchik, Frank, '40..1831 Barnum av., Bridgeport 8, Conn. ALR
②Turnbull, F. M., '16
Wilshire Med. bldg., Los Angeles 5, Calif. ALR
①Turner, H. H., '14.....Clark bldg., Pittsburgh 22, Pa. OP
①Turner, Oliver W., '09.....37 Stone st., Augusta, Maine OALR
Turnley, William H., '33...1 Atlantic st., Stamford, Conn. ALR
Tusak, Ervin A., '31....115 E. 90th, New York 28, N. Y. OP
Twitchell, Emmus G., '37.....266 Main, Burlington, Vt. OALR
Tygett, Glenn J., '39
215 N. Sprigg st., Cape Girardeau, Mo. ALR
Tyler, Richard E., '43, The Norwood Clinic,
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Tyson, John McClure, '40.....3 S. Brady st., DuBois, Pa. ALR

U

Uhler, S. Mann, '30.....104 N. 8th, Allentown, Pa. ALR
Ulmer, David H. B., '39..199 Chestnut, Moorestown, N. J. ALR
Underwood, John H., '38.....Brant bldg., Canton 2, Ohio ALR
Unsworth, Arthur C., '38, Lt. Col. (M.C.),
709 N. Greenway Dr., Coral Gables 34, Fla. OP
①Upham, Helen F., '07..305 Third av., Asbury Park, N. J. OALR
Usen, Louis, '36....278 New York av., Brooklyn 16, N. Y. ALR

V

Vadala, Anthony J., '38, Lt. Col. (M.C.),
Station Hospital, Fort Totten, N. Y. OALR
Vail, Derrick, '31, Col. (M.C.),
A.P.O. 871, New York, N. Y. OP
Vaile, Ivan R., '36.....384 Bloor st. W., Toronto, Can. ALR

Valone, Frank H., '31.....107 W. Court, Rome, N. Y. ALR
Van Alyea, O. E., '35.....135 S. LaSalle, Chicago 3, Ill. ALR
Van Buskirk, Edmund Linford, '41
308-312 N. 8th, Lafayette, Ind. OP
②Van der Hoeve, Prof. J., '21
Rijnsburgerweg 6A, Leiden, Holland
Vandevere, William E., '32
1st Nat'l Bank bldg., El Paso, Texas OALR
Vann, Homer King, '41
410 Cedar st. N. W., Washington 12, D. C. ALR
Van Osdol, Harry A., '31
Chamber of Commerce, Indianapolis 4, Ind. ALR
Van Poole, G. McD., '24..Young bldg., Honolulu 9, Hawaii, OALR
Van Wien, Stefan, '43, Lt. (M.C.), 0-496435,
A.P.O. 813, c/o Postmaster, New York OP
Vanzant, Thomas J., '38, Lt. (M.C.), Station Hosp.
Keesler Field, Biloxi, Miss. OP
Vaughn, Benjamin H., '38
Adams bldg., Port Arthur, Texas ALR
②Vaughn, E. H., '19
Peoples Nat'l Bank bldg., Tyler, Texas OALR
Veach, Oscar Lloyd, '40
Whitney Trust bldg., Sheridan, Wyo. OP
①Veasey, C. A., Sr., '06
Paulsen Med. and Dental bldg., Spokane 8, Wash. OP
Veasey, C. A., Jr., '27
Paulsen Med. and Dental bldg., Spokane 8, Wash. OALR
Veirs, Everett Raymond, '40, Major (M.C.),
Lawson General Hopsital, Atlanta, Ga. ALR
Veitch, Ambert H., '37.....170 St. George, Toronto, Can. ALR
Verhoeff, F. H., '36
395 Commonwealth av., Boston 15, Mass. OP
Vermeren, John C., '38
Lt. Comdr. (M.C.), 4753 Broadway, Chicago 40, Ill. ALR
Vermillion, J. S., '41, Major (M.C.),
A.P.O. 680, c/o P. M., New York, N. Y. ALR
Victor, Karl N., '39
1010 Brown bldg., 321 W. Broadway, Louisville 2, Ky. ALR
Vogel, Felix P. S., '36
1749 Grand Concourse, New York 53, N. Y. ALR
Vogt, George C., '37.....140 Chapin, Binghamton, N. Y. ALR
①Voigt, C. B., '12.....1702 Broadway, Mattoon, Ill. OALR
Volk, Benjamin M., '38
146 Washington av., Albany 6, N. Y. ALR
Von der Heydt, Robert, '26
25 E. Washington, Chicago 2, Ill. OP
von Lackum, John K., '37
Mer. Nat'l Bank bldg., Cedar Rapids, Iowa ALR
von Zelinski, Walter Franz, '41, Col. (M.C.),
Headquarters Fort Custer, Michigan OP
Voorhees, Earle W., '35
8 Garfield place., Poughkeepsie, N. Y. ALR
Vorisek, Elmer A., '33, Major (M.C.),
8 E. Shadowlawn av. N. E., Atlanta, Ga. OP
Vossler, Albert E., '37
David Whitney bldg., Detroit 26, Mich. OALR
Votaw, Robert E., '37, Maj. (M.C.), Station Hospital,
Majors Army Air Field, Greenville, Texas ALR
Vrooman, Carl O., '40, Maj. (M.C.), Bradley Field
Station Hospital, Windsor Locks, Conn. OP

W

Wachsberger, Alfred, '31, Major (M.C.),
Hammond General Hospital, Modesto, Calif. ALR
Wagener, Henry P., '26.....Mayo Clinic, Rochester, Minn. OP
Wagers, Arthur J., '20....1429 Spruce, Philadelphia 2, Pa. ALR
Wagner, W. A., '27...200 Carondelet, New Orleans 12, La. ALR
Wails, Theodore G., '38
Medical Arts bldg., Oklahoma City 2, Okla. ALR
Waldapfel, Richard, '40
De Merschman Gardens, Grand Junction, Colo. ALR
Waldeck, E. A., '34
238 W. Wisconsin av., Milwaukee 3, Wis. OALR
Waldman, Joseph, '37, Maj. (M.C.), Park Manor Apts.,
4000 Girard av., Philadelphia 4, Pa. OP
②Walker, C. E., '18.....1732 High, Denver 6, Colo. OP
Walker, Charles E., Jr., '37, Major (M.C.),
1732 High st., Denver 6, Colo. OP
Walker, George W., '32
T. W. Patterson bldg., Fresno 1, Calif. ALR
Walker, John R., '32.T. W. Patterson bldg., Fresno 1, Calif. OP
Walker, O. J., '35.Home S. & L. bldg., Youngstown 3, Ohio ALR
Walker, Oliver Frank, '42
2873 Lewiston road, Niagara Falls, N. Y. ALR
Walker, R. M., '40, Lt. (M.C.), U.S.N.R.,
201 N. Main st., Sumter, S. C. ALR
Wall, John W., '42, Lt. (M.C.), U.S.N.R.,
c/o F. P. O., San Francisco, Calif. ALR
Wallerstein, Emanuel U., '41
Professional bldg., Richmond 19, Va. ALR
Wallner, Linden J., '38, Major (M.C.),
4350 Bobolink Terr., Skokie, Ill. ALR
Walls, Edward G., '35, Lt. Comdr. (M.C.),
Fleet P.O., San Francisco, Calif. ALR
Walsh, Theodore Edwin, '40
640 S. Kingshighway blvd., St. Louis 10, Mo. ALR
Walsh, Thomas J., '39...2059 Parrish, Philadelphia 30, Pa. ALR
Waltz, Harold Darnell, '42, Major (M.C.),
Station Hospital, Perrin Field, Sherman, Texas ALR
Ward, J. W., '30.....2607 Lee, Greenville, Texas ALR
Warner, Raymond C., '38
324 E. Wisconsin av., Milwaukee 2, Wis. OP
Warner, Robert J., '29.....706 Church, Nashville, Tenn. OP
Warren, William C., Jr., '28..Doctors bldg., Atlanta 3, Ga. ALR
Washick, Frank, '42.501 Cottman st., Philadelphia 11, Pa. ALR
Watke, Frederic Martin, '41
Medical Arts bldg., Omaha 2, Nebr. ALR
Watkins, H. R., '30....Greiesheim bldg., Bloomington, Ill. ALR
Watkins, J. O., '41, Major (M.C.),
McCloskey General Hospital, Temple, Texas OP
Watnick, Joseph Louis, '41
10 W. 96th, New York 25, N. Y. ALR
②Watson, H. de Witt, '15...151 Front, Binghamton, N. Y. OALR
Watson, O. Alton, '37
1200 N. Walker, Oklahoma City 3, Okla. ALR
Wattles, F. Merrill, '39, Lt. Comdr. (M.C.),
U. S. Naval Hospital, Key West, Fla. ALR
②Waugh, J. M., '19
Ridge Road, R.F.D. No. 1, Willoughby, Ohio OALR
Weaver, Delmar Franklin, '41
Henry Ford Hospital, Detroit 2, Mich. ALR
Weaver, Emerson M. F., '43, Major (M.C.), 0-263171,
A.P.O. 951, c/o Postmaster, San Francisco, Calif. OP
Weaver, Homer V., '32
625 Cleveland av. N. W., Canton 3, Ohio ALR
①Weaver, T. W., '14.....201 N. Main, Wichita 2, Kan. OP
Webb, M. A., '43.....4500 Olive st., St. Louis 8, Mo. ALR
Weber, H. C., '28, Capt. (M.C.),
U.S.N., 1112 Mason av., Drexel Hill, Pa. OALR
Weber, Julius A., '39.....Stimson bldg., Seattle 1, Wash. ALR
Webster, David H., '37...140 E. 54th, New York 22, N. Y. OP
Webster, F. R., '30...State Tower bldg., Syracuse 2, N. Y. OP

Wechsler, A. H., '34
340 New York av., Brooklyn 13, N. Y. **ALR**
①Weeks, John E., '04, (Retired),
360 Edgecliff rd., Portland 1, Ore. **OP**
Weeth, C. R., '30.....37 Remsen, Brooklyn 2, N. Y. **ALR**
Wegner, Edwin A., Jr., '38, Capt. (M.C.),
Station Hospital, Camp Shanks, Orangeburg, N. Y. **ALR**
Weidlein, Ivan F., '30
10515 Carnegie av., Cleveland 6, Ohio **ALR**
Weih, E. P., '20.....217 Fifth av. S., Clinton, Ia. **OALR**
②Weil, A. I., '16....3439 Prytania, New Orleans 15, La. **ALR**
Weinberg, Herman Charles, '42, Lt. (M.C.), 0-496859,
A.P.O. 871, c/o P. M., New York, N. Y. **OP**
Weinman, Edward B., '39, Capt. (M.C.),
Foster General Hospital, Jackson 115, Miss. **OP**
Weinstein, M. A., '27....1530 Locust, Philadelphia 2, Pa. **ALR**
Weinstein, Philip, '39..400 Ingraham bldg., Miama 32, Fla. **ALR**
Weinstein, Samuel, '37, 441 Ocean av., Brooklyn 26, N. Y. **ALR**
Weiss, Harry, '40, Capt. (M.C.),
140-50 Ash av., Flushing, N. Y. **OP**
Weiss, J. Allan, '32, Lt. Comdr. (M.C.), U. S. Naval
Con. Training Center, B. O. Q.-04, Davisville, R. I. **ALR**
Weisser, Charles William, '42, Capt. (M.C.),
76th General Hospital, Vancouver, Washington **OP**
②Weisser, E. A., '17.....May bldg., Pittsburgh 22, Pa. **OP**
Weizenblatt, Sprinza, '38
New Medical bldg., Asheville, N. C. **OP**
Welch, Robert S. G., '22, 86 State Circle, Annapolis, Md. **OALR**
Weldon, Wm. A., '33.....Glasgow, Ky. **OP**
Wellford, B. R., '43
510 Medical Arts bldg., Richmond 19, Va. **ALR**
①Wells, David W., '08, 31 Bay State rd., Boston 15, Mass. **OP**
Welsh, Thomas F., '34
First Nat'l Bank bldg., Salt Lake City 1, Utah **OALR**
Welt, Bernard, '43, 135 New York av., Brooklyn 16, N. Y. **ALR**
Wenaas, Elmer J., '38....24 Wick av., Youngstown, Ohio **OP**
Wencke, Carl G., '25
1015 Security Tower, Battle Creek, Mich. **ALR**
Wendel, J. S., '23. David Whitney bldg., Detroit 26, Mich. **ALR**
Wenner, W. T., '30....St. Mary's bldg., St. Cloud, Minn. **OP**
Wescott, Virgil, '35....30 N. Michigan av., Chicago 2, Ill. **OP**
Wesson, Thomas W., '43, 124 S. E. 1st., Evansville 8, Ind. **ALR**
Westervelt, H. O., '32
239 Pipestone, Benton Harbor, Mich. **ALR**
②Westlake, Samuel B., '18
Beaumont Med. bldg., St. Louis 8, Mo. **ALR**
Wetzel, John O., '30..Olds Tower bldg., Lansing 8, Mich. **OP**
Wexler, David, '30.....103 E. 86th, New York 28, N. Y. **OP**
Weymuller, Ernest A., '36
Capt. (M.C.), Station Hosp., Mitchell Field, L.I., N. Y. **ALR**
Whalen, Edward J., '28.....750 Main, Hartford 3, Conn. **ALR**
Whalman, Harold Frederic, '40
R. 1147, 727 W. 7th st., Los Angeles 14, Calif. **OP**
Wheeler, Maynard C., '35, 30 W. 59th, New York 19, N. Y. **OP**
Wheeler, Merritt W., '23..Lowry bldg., St. Paul 2, Minn. **OALR**
Whelan, G. L., '25.....2100 Walnut, Philadelphia 3, Pa. **ALR**
Whelan, William F., '34..2004 Walnut, Philadelphia 3, Pa. **ALR**
①Whisnant, A. M., '13
Professional bldg., Charlotte 2, N. C. **OALR**
②Whitaker, Joel, '17
807 Hume-Mansur bldg., Indianapolis 4, Ind. **OALR**
White, Carl Haper, '42, Lt. Comdr. (M.C.), Naval
Aviation Base, Minneapolis 6, Minn. **ALR**
White, Clarence Hunt, '42.....Box 257, Henderson, N. C. **ALR**
White, Francis W., '37..471 Park av., New York 22, N. Y. **ALR**
White, Herbert T., '37.....National bldg., Flint 3, Mich. **OP**
White, James W., '34....15 Park av., New York 16, N. Y. **OP**
White, J. Warren, '37, Medical Arts bldg., Norfolk 10, Va. **OALR**
White, Vivian P., '31
Tribune Telegram bldg., Salt Lake City 1, Utah **ALR**
Whitney, E. L., '30..Henry Ford Hosp., Detroit 2, Mich. **OP**
Whitney, P. T., '36.....390 Main, Worcester 8, Mass. **OP**
Whitsell, Fay M., '37, Capt. (M.C.),
Ashburn General Hospital, McKinney, Texas **OP**
Whitworth, John Ewing, '41.....49 Hammond, Bangor, Me. **ALR**
Wible, LeRoy E., '35, Lt. Comdr. (M.C.),
Port Deposit, Bainbridge, Md. **ALR**
Wickliffe, Thompson F., '31....Tweedy bldg., Jasper, Ala. **ALR**
①Wiener, Alfred, '08..550 Park av., New York 21, N. Y. **OALR**
①Wiener, Meyer, '12
Box 685, 321 Alameda blvd., Coronado, Calif. **OP**
Wieselthier, Maurice L., '42
1322 Union st., Brooklyn 13, N. Y. **OP**
Wiest, Philip R., '43, Capt. (M.C.),
Station Hospital, Selfridge Field, Mich. **ALR**
Wiggin, William I., '29.....202 Central, Lowell, Mass. **ALR**
Wilber, Inez Edith, '40
1746 K st. N. W., Washington 6, D. C. **OP**
①Wilbur, Edward P., '03
American Nat'l Bank bldg., Kalamazoo 4, Mich. **OALR**
Wilcox, Malcolm B., '43....5 West 21st st., Kerney, Nebr. **ALR**
Wiley, Jason L., '39.....Metcalf bldg., Auburn, N. Y. **OP**
Wilkerson, Wm. W., Jr., '32
Bennie-Dillon bldg., Nashville 3, Tenn. **ALR**
Wilkins, Ellis LeRoy, '42
McWilliams bldg., Clarksdale, Miss. **ALR**
Wilkins, Samuel H., '34
270 Commonwealth av., Boston 16, Mass. **OP**
Wilkinson, A. P., '30
3001 W. Grand blvd., Detroit 2, Mich. **ALR**
Wilkinson, Joel A., '37
1130 Louisiana av., Shreveport 7, La. **OALR**
Wilkinson, Oscar, '30
1408 L st. N. W., Washington 5, D. C. **OALR**
Wilkinson, Richard W., '34
1408 L st. N. W., Washington 5, D. C. **ALR**
Wilkinson, Wallace B., '39..521 E. 10th, Dallas 8, Texas **ALR**
Williams, Henry L., '37....Mayo Clinic, Rochester, Minn. **ALR**
Williams, Horace J., '22
5908 Greene, Germantown, Philadelphia 44, Pa. **ALR**
Williams, John W., '43, Capt. (M.C.), General Dispensary
7 Ser. Com., 18 floor, Continental bldg., St. Louis 8, Mo. **ALR**
Williams, M. M., '27..Medical Arts bldg., Scranton 3, Pa. **OP**
Williams, Mortimer H., '39, Lt. Comdr. (M. C.),
Navy 168, c/o Fleet P. O., New York, N. Y. **ALR**
Williams, Robert Judson, '41, Lt. Comdr. (M.C.),
Naval Air Station Disp., Sanford, Fla. **ALR**
Williams, Russell I., '43, 422 Hynds bldg., Cheyenne Wyo. **ALR**
Williams, Thomas J., '37..1604 Chicago av., Evanston, Ill. **ALR**
①Williams, W. C., '08.....Jefferson bldg., Peoria 2, Ill. **OALR**
Williams, Walton H., '21, Castell bldg., Middletown, Ohio **OALR**
Willison, Eugene E., '39
Merced Clinic bldg., Merced, Calif. **OP**
Willson, Wesley W., '28, Lt. Col. (M.C.),
Station Hosp., Camp Haan, Calif. **OALR**
Wilmes, Malcolm N., '43, Major (M.C.),
APO 555, c/o P.M., New York, N. Y. **ALR**
Wilson, Alfred, '39, Maj. (M.C.),
1518 20th av., Gulfport, Miss. **OP**
Wilson, Alpheus K., '38
334 St. James bldg., Jacksonville 2, Fla. **ALR**
Wilson, Clinton A., '40, Major (M.C.)
951 S. Kenisto av., Los Angeles 6, Calif. **OP**
Wilson, Frank E., '33.....302 State, New London, Conn. **ALR**

Wilson, George E., '38....108 Main, Saranac Lake, N. Y. **ALR**
Wilson, Harold A., '41, 71 Maiden Lane, Kingston, N. Y. **ALR**
Wilson, Hudson J., '36
1st National Bank bldg., Ithaca, N. Y. **ALR**
Wilson, Otis M., '42.....502 3rd st., Wausau, Wis. **OP**
Wilson, Paul E., '39.....408 Franklin, Clarksville, Tenn. **OP**
Wilson, Robert G., '31....304 East Main, Batavia, N. Y. **ALR**
Wilson, William Hildebrand, '42, Capt. (M.C.),
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1882 Grand Concourse, New York 57, N. Y. **OP**
Windham, R. E., '28
234 W. Beauregard blvd., San Angelo, Texas **OALR**
Winer, Albert S., '43
1835 Eye St., N. W., Washington 6, D. C. **ALR**
Winger, Ira B., '27.....709 Madison av., Toledo 2, Ohio **OALR**
Winkler, Herman, '34....224 Thayer, Providence 6, R. I. **ALR**
Winn, Thomas M., '39, 129½ Maple av., Covington, Va. **OALR**
Winn, William R., '38.....Dakota Clinic, Fargo, N. D. **ALR**
Winston, Julius, '36.....2021 Spruce, Philadelphia 3, Pa. **ALR**
Winter, George E., '22
117 Michigan av. W., Jackson, Mich. **OALR**
Winter, John A., '23.....205 W. 2nd, Duluth 2, Minn. **OALR**
①**Wise, Ralph C.**, '14.....News bldg., Mansfield, Ohio **OALR**
Wiser, Henry Joseph, '41, Comdr. (M.C.),
Fleet P. O., San Francisco, Calif. **ALR**
Wishart, David Edmund Staunton, '42
Medical Arts bldg., Toronto 5, Can. **ALR**
Wojniak, Frank, '43, 4649 S. Ashland av., Chicago 9, Ill. **ALR**
Wolcott, Charles C., '31.....27 Ludlow, Yonkers, N. Y. **ALR**
Wold, K. C., '24
Lowry Med. Arts bldg., St. Paul 2, Minn. **OALR**
Wolf, Lewis R., '43
3474 Frankford av., Philadelphia 34, Penn. **OP**
Wolfe, Hugh C., '37....102 N. Elm, Greensboro, N. C. **ALR**
②**Wolfe, Otis R.**, '19
Masonic Temple, Marshalltown, Iowa **OP**
Wolfe, William C., '41, Capt. (M.C.), Army and Navy
General Hospital, Hot Springs, Ark. **ALR**
Wolpaw, Benjamin J., '36, Lt. Comdr. (M.C.), Naval
Air Station Dispensary, Pensacola, Fla. **OP**
Womack, David R., '33, Comdr. (M.C.), USNR,
c/o Fleet P. O., San Francisco, Cal. **ALR**
Wood, Earl L., '28, Col. (M.C.),
Station Hospital, Camp Anza, Arlington, Cal. **ALR**
Wood, Edward C., '35, Major, (M.C.),
Sta. Hosp., Charleston A.A.B., Charleston, S. C. **OP**
Wood, Everet H., '40, Capt. (M.C.),
A.P.O. 957, c/o P. M., San Francisco, Calif. **OP**
Wood, Spencer, '37.....500 Verret, New Orleans 15, La. **ALR**
Wood, V. Visscher, '28
Beaumont Med. bldg., St. Louis 8, Mo. **ALR**
Woodman, Degraaf, '33, 156 E. 37th, New York 16, N. Y. **ALR**
Woodrow, Jack H., '41, Capt. (M.C.),
A.P.O. 143, c/o Postmaster, New York, N. Y. **ALR**
①**Woodrow, Frederick E.**, '13
Metropolitan bldg., St. Louis 3, Mo. **OP**
Woodruff, George H., '32....250 N. Ottawa, Joliet, Ill. **ALR**
①**Woodruff, H. W.**, '11.....250 N. Ottawa, Joliet, Ill. **OALR**
Woodry, Norman L., '36..10 Witherell, Detroit 26, Mich. **ALR**
Woods, Alan C., '39
Johns Hopkins Hospital, Baltimore 5, Md. **OP**
②**Woods, R. H.**, '15.....153 Marquette, La Salle, Ill. **OP**
Woodson, B. Palmer, '28
Scott and White Hospital, Temple, Texas **OALR**
Woodson, Warren B., '33
601-22 Professional bldg., Temple, Tex. **OALR**
Woodward, F. D., '29
104 E. Market, Charlottesville, Va. **ALR**
Woodward, John F., Jr., '36, Lt. Comdr. (M.C.),
Navy 231, Fleet Post Office, New York, N. Y. **ALR**
Woodward, Paul Maxwell, '41
4140 Hamilton av., Cincinnati 23, Ohio **ALR**
Woodworth, Wm. P., '27
2501 W. Grand blvd., Detroit 8, Mich. **OALR**
Woolfolk, William L., '43, 115 E. 4th st., Owensboro, Ky. **ALR**
Woolsey, William Jinkens, '42
212 N. 7th., Waco, Texas **OP**
Worcester, George F., '41....220 Engle, Englewood, N. J. **ALR**
Work, Walter P., '43, Capt (M.C.),
O'Reilly General Hospital, Springfield, Mo. **ALR**
Wotring, John M., '39.....220 N. 6th, Reading, Pa. **OP**
Wright, Edward S., '38, Lt. Comdr. (M.C.), USNR, Naval Hosp.,
Naval Air Training Center, Pensacola, Fla. **ALR**
①**Wright, J. Wm.**, '14
301 Hume-Mansur bldg., Indianapolis 4, Ind. **ALR**
Wright, R. Percy, '32, Col. (R.C.A.M.C.),
6065 Sherbrook West, Montreal, Can. **ALR**
Wright, Walter W., '26
Medical Arts bldg., Toronto 5, Can. **OP**
①**Wurtz, W. J. M.**, '13..333 Linwood av., Buffalo 9, N. Y. **ALR**
①**Wylie, C. B.**, '10.....293 High, Morgantown, W. Va. **OALR**

Y

Yavelow, Charles S., '41
244 Bedford av., Mt. Vernon, N. Y. **ALR**
Yazel, Herman E., '20..Rialto bldg., Kansas City 6, Mo. **OALR**
Yinger, Starling C., '41
First Nat'l Bank bldg., Springfield, Ohio **ALR**
Yost, Walter M., '28.....Lake av., Greenwich, Conn. **OP**
Young, Charles A., '26, Comdr. (M.C.),
U. S. Naval Hospital, Bainbridge, Maryland **OP**
Young, Clarence M., '35.....Box 1506, Medford, Ore. **ALR**
Youngerman, William M., '38, Major (M.C.),
Station Hosp., Lowry Field, Denver, Col. **ALR**
Youngs, N. A., '36
Grand Forks Clinic, Grand Forks, N. D. **ALR**
Yudkin, Arthur M., '37, 257 Church, New Haven 10, Conn. **OP**

Z

Zacks, Myron A., '39..1737 Chestnut, Philadelphia 3, Pa. **ALR**
Zbar, Joseph E., '42, 90 Duncan av., Jersey City, N. J. **ALR**
②**Zehnder, A. Charles**, '19
188 Roseville av., Newark 7, N. J. **OP**
Zekman, Theodore N., '38, Lt. (M.C.),
1627 Hyde Park Blvd., Chicago, Ill. **OP**
Zeman, Michael S., '43, Capt. (M.C.),
296 North Quaker Lane, Hartford, Conn. **ALR**
②**Zemar, S. G.**, '17.....Stuart bldg., Lincoln 8, Nebr. **OALR**
Zentmayer, Wm., '21..1930 Chestnut, Philadelphia 3, Pa. **OP**
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Zinkhan, A. M., '20
1801 Eye st. N. W., Washington 6, D. C. **ALR**
Zinn, Waitman Farnsworth, '40
Medical Arts bldg., Baltimore 1, Md. **ALR**
Zubak, Matthew F., '28
Citizens Mut. Trust bldg., Wheeling, W. Va. **OALR**
Zuckerman, Joshua, '36..875 Park av., New York 21, N. Y. **OP**
Zwerling, Samuel, '36
789 St. Mark's av., Brooklyn 13, N. Y. **ALR**

Geographical

ALABAMA

BIRMINGHAM
Constantine, K. W.....2820 Berwick rd., Z5 OP
Fisher, Gilbert Eugene.....Medical Arts bldg., Z5 ALR
Hardy, Walter B.....2121 Highland av., Z5 ALR
Pope, Ernest C.....2021 Sixth av. N., Z3 ALR
Rucker, E. W., Jr.....Woodward bldg., Z3 ALR
Tyler, Richard E.
The Norwood Clinic, 2501 N. 16th av., Z4 ALR

CAMP RUCKER
Sacheroff, Louis, Capt. (M.C.).....Station Hospital ALR

DOTHAN
Keyton, John A.....304 N. Oates ALR

GADSDEN
Frank, Herman W.....Medical bldg. OP
Isbell, Euclid A.....948 Forrest av. ALR

HUNTSVILLE
Lazar, A. M., Maj. (M.C.).....407 Eastis st. ALR

JASPER
Wickliffe, T. F.....Tweedy bldg. ALR

MOBILE
Perdue, James D.....P. O. Box 514, Z4 OP
Sanders, J. Gillis...714 Merchants Nat'l Bank bldg., Z11 ALR

MONTGOMERY
Hagood, Daniel S.....822 First Nat'l Bank bldg., Z4 OP
Holding, Bruce F.....Bell bldg., Z4 ALR
Nodine, Edward R.....1110 Bell bldg., Z4 ALR
Thigpen, C. A.....401 S. Court, Z5 OALR

SELMA
Evans, John D., Maj. (M.C.),
Station Hospital, Craig Field ALR

TUSCALOOSA
Searcy, H. B.....2501 Sixth OALR

ARIZONA

CHANDLER
Henton, George H.....Lt. Col. (M.C.), Williams Field OP

PHOENIX
Brinkerhoff, David E.
Lt. Comdr. (M.C.), 320 Professional bldg. ALR
Case, Paul H.....15 E. Monroe OP
Cruthirds, Archie Edward.....Professional bldg. ALR
Melton, B. L., Jr.....15 E. Monroe, OP
Robb, Mayo.....Professional bldg. ALR
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Swetman, C. R. K.....219 S. Mt. Vernon ALR

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Brown, Earl Henderson.....130 S. Scott ALR.

ARKANSAS

BLYTHEVILLE
Scales, W. M.....Maj. (M.C.), Station Hospital, BAAF OP

EL DORADO
Russell, Martin V.....200 W. Elm st. ALR

FORT SMITH
Moulton, Everett C.....Merchants National Bank bldg. OALR
Moulton, Herbert.....Merchants National Bank bldg. OALR

HOT SPRINGS
Cunningham, Bernard P.
Capt. (M.C.), Army-Navy General Hospital ALR
Goldwaite Ralph H., Brig. Gen. (M.C.),
Army and Navy General Hospital OALR
King, O. H.....Medical Arts bldg. OALR
Noe, Carl A., Maj. (M.C.),
Army and Navy General Hospital OALR
Wolfe, William C., Capt. (M.C.),
Army and Navy General Hospital ALR

LITTLE ROCK
Caldwell, Robert.....New Donaghey bldg. OALR
Cosgrove, Kingsley White.....115 East Capitol OALR
Mahoney, Paul Leo.....Donaghey bldg. ALR
Moore, Robert B.....2000 Main ALR

NEWPORT
Marks, Moses I...Capt. (M.C.), Newport Army Air Field ALR

PINE BLUFF
Payne, Virgil LeRoy.....Simmons National bldg. ALR

WALNUT RIDGE
Paulk, James R.
Capt. (M.C.), Station Hospital, Army Air Field OP

CALIFORNIA

ALAMEDA
Smithies, Harold R.....2241 Central av. ALR

ALHAMBRA
Johnson, Ernest D.....317 W. Main OALR

ARLINGTON
Morris, Samuel A., Capt. (M.C.),
Station Hospital, Camp Anza OP
Wood, Earl L. Col. (M.C.), Station Hospital, Camp Anza ALR

BAKERSFIELD
Donohoe, Edward C., Lt. Col. (M.C.),
Station Hospital, A.A.F.P.S. (B), Minter Field ALR

BERKELEY
Knight, John S., Lt. Comdr. (M.C.),
U.S.N.R., 162 Hillcrest rd., Z5 ALR

CALIFORNIA — Continued

BEVERLY HILLS

Cooperman, Harold O., Suite 210, Beverly Prof. bldg., 9615 Brighton Way **OP**
Dow, Julian Neal....9730 Wilshire blvd. **OP**
Holden, Harold M....514 N. Linden dr. **ALR**
Lemere, H. B....9615 Brighton Way **OALR**
Mayer, Leo L....Suite 204, 415 N. Camden dr. **OP**
Romonek, Philip L....415 N. Camden dr. **OALR**
Shumaker, Phil W....9730 Wilshire blvd. **ALR**
Semenov, Herman....Maj. (M.C.), 415 N. Camden dr. **ALR**
Wimmer, Shirley D....153 S. Lasky dr. **ALR**

BURBANK

Monaco, Louis....233 S. San Fernando blvd. **ALR**

CAMP COOKE

Priest, R. E....Capt. (M.C.), Station Hospital **ALR**

CAMP HAAN

Willson, W. W., Lt. Col. (M.C.)....Station Hospital **OALR**

CAMP STONEMAN

Cheney, Ralph E....Major (M.C.), Station Hospital **OP**
Culpepper, William S....Col. (M.C.), Station Hospital **ALR**

CAMP TORRENCE

MacPherson, William, Capt. (M.C.), 205 Hospital Ship Complement, c/o Station Hosp. **OP**

CORONADO

Hardy, Guerdan....Lt. Comdr. (M.C.), 960 G av. **OALR**
Wiener, Meyer....Box 685, 321 Alameda blvd. **OP**

COVINA

Kremen, Abraham....Capt. (M.C.), 514 W. Rowland av. **OP**

EL CENTRO

Edwards, S. R....107 S. Fifth **OALR**

EL TORO

Boyd, G. G....Lt. Comdr. (M.C.), Marine Air Station **ALR**

FRESNO

Irvine, S. R..Capt. (M.C.), Station Hosp., Hammer Field **OP**
Olson, George W....1759 Fulton, Z1 **ALR**
Piszkiewicz, Frank J. Major (M.C.), Station Hospital, Hammer Field **ALR**
Trowbridge, Dwight H....T. W. Patterson bldg., Z1 **OP**
Walker, George W....T. W. Patterson bldg., Z1 **ALR**
Walker, John R....T. W. Patterson bldg., Z1 **OP**

GLENDALE

Trott, Leslie De Nyse....143 North Brand, Z3 **ALR**

HAMILTON FIELD

Howell, Homer P....Capt. (M.C.), Sta. Hosp. **OP**

HOLLYWOOD

James, Voyle....6253 Hollywood blvd. **ALR**
Naftzger, J. B....6777 Hollywood blvd., Z28 **ALR**

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Kinkade, Joseph M....359 E. Manchester blvd. **ALR**

LAGUNA BEACH

Neher, Edwin M....475 El Camino, Del Mar **OP**

LA JOLLA

Seidel, U. D....447 Belvedere st. **ALR**

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Sherman, A. Edward Lt. Col. (M.C.) (Retired), Rt. 1, Box 1401 **OALR**

LOMA LINDA

Schroeder, Milo Carl....Loma Linda Sanitarium **ALR**

LONG BEACH

Albright, Paul M. Capt. (M.C.), U. S. Naval Hosp. **OALR**
Brandenburg, Kenneth C....110 Pine av., Z2 **OALR**
Miller, A. H. Lt. (M.C.), U.S.N.R., U. S. Naval Hosp. **ALR**
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Thornburgh, Robert G....Professional bldg., Z2 **OP**

LOS ANGELES

Abraham, Samuel V....1930 Wilshire blvd., Z5 **OP**
Albaugh, Clarence H....727 W. 7th (suite 1131), Z14 **OP**
Bourbon, Oliver P....6561 Meridian st., Z42 **OALR**
Boyce, William A....Roosevelt bldg., Z14 **OP**
Brown, J. M....1136 W. 6th, Z14 **ALR**
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Colver, Benton Noble....436 S. Boyle av., Z33 **ALR**
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Dayton, G. O....1401 S. Hope, Z15 **OALR**
Donoher, William D....635 S. Westlake av., Z5 **OALR**
Ellis, Orwyn H., Lt. (j. g.) (M.C.), 647 Warner av., Z24 **OP**
Fairchild, Nora M....939 S. Figueroa, Z15 **OP**
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Goldberg, Sylvan S....1917 Wilshire blvd., Z5 **ALR**
Goodhill, Victor 1101 Wilshire Medical bldg., 1930 Wilshire blvd., Z5 **ALR**
Gosney, Charles W....5217 Hollywood blvd., Z27 **OP**
Gundrum, Lawrence Kramer....1930 Wilshire blvd., Z5 **ALR**
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House, Howard Payne....511 S. Bonnie Brae, Z5 **ALR**
House, Leland Richmond....312 N. Boyle av., Z33 **ALR**
Howarth, Ervin McIntyre c/o Mrs. M. Verhusen, 4126 W. 61st st. **ALR**
Irvine, A. Ray....Room 700, 727 W. 7th st., Z14 **OP**
Jesberg, Simon....500 S. Lucas av., Z13 **ALR**
Kully, B. M....3875 Wilshire blvd., Z5 **ALR**
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McLaughlin, R. C....3780 Wilshire blvd., Z5 **ALR**
Maghy, Charles A....Wilshire Med. bldg., Z5 **OP**
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Rubin, Henry J., Lt. (M.C.), 611 N. Stanley av., Z36 **ALR**
Shmukler, B. Cecelia....1930 Wilshire blvd., Z5 **OP**
Turnbull, F. M....Wilshire Medical bldg., Z5 **ALR**
Whalman, Harold Frederic ....1147-727 W. 7th, Z14 **OP**
Wilson, Clinton A., Maj. (M.C.), 951 S. Keniston av., Z6 **OP**

CALIFORNIA — Continued

MARCH FIELD
Harris, E. W.....Maj. (M.C.), Station Hospital ALR

MARYSVILLE
Lewis, J. D.....725 Fourth Street OALR

MENLO PARK
Snydacker, Daniel, Major (M.C.), Dibble General Hosp. OP

MERCED
Willison, Eugene E.....Merced Cline bldg. OP

MODESTO
Cattles, Newell H., Capt. (M.C.), Hammond Gen. Hosp. OALR
Wachsberger, Alfred, Maj. (M.C.), Hammond Gen. Hosp. ALR

MONTEREY
Clark, Howard E.....576 Hartnell st. OALR

OAKLAND
Barbour, Fleming A., Lt. (M.C.), U. S. Naval Hosp., Z14 OP
Keeler, Nelson S.....411 Thirtieth, Z9 ALR
McAlester, Andrew Walker, 3rd
Lt. Comdr. (M.C.), Naval General Hosp., Z14 OP
McClure, George.....411 Thirtieth, Z9 ALR
Nutting, Raymond J.....411 Thirtieth, Z9 OP

OCEANSIDE
Mulligan, Harold R., Comdr. (M.C.), 2028 S. Pacific ALR
Schenck, Harry P., Comdr. (M.C.)
U. S. Naval Hosp., Santa Margarita Ranch ALR

ONTARIO
Hooval, John Harold .....105 W. C st. ALR

PALM SPRINGS
Chapman, Vernon A.....435 N. Palm Canyon drive OALR

PALO ALTO
Sewell, E. C.....430 El Escarpado ALR

PASADENA
Allen, J. H.....2081 Garfias Drive, Z7 ALR
Cady, D. W.....65 N. Madison av., Z1 OP
Darrow, C. H.....3020 E. Colorado st., Z8 ALR
Decker, Russell M.....65 N. Madison av., Z1 ALR
Dysart, Ben Robnett.....65 N. Madison av., Z1 ALR
Henninger, L. L.....65 N. Madison av., Z1 OALR
Henry, L. L.....595 E. Colorado st., Z1 OP
Johnson, Henrietta May.....595 E. Colorado, Z1 OP
Miller, Theodore E.....First Trust bldg., Z1 ALR
Reed, James Ross.....201 N. El Molino av., Z4 OP
Risser, E. D.....423 First Trust bldg., Z1 OALR
Roberts, William H.....65 N. Madison av., Z1 OP
Scanlon, Wilko Gruver.....201 N. El Molino av., Z4 ALR.

POMONA
Roberts, Gilbert Jay
Lt. Comdr. (M.C.), 586 N. Main st. ALR

PORT HUENEME
Greenlee, Max R., Lt. Comdr. (M.C.),
Acorn 45, Maritime bldg. ALR

SACRAMENTO
Spencer, George Albert.....1025 Ninth, Z14 OALR
Tillotson, R. S.....Medico-Dental bldg., Z14 ALR

SAN BERNARDINO
Penta, John J., Major (M.C.), 01695906
Station Hospital S.B.A.A.F. ALR

SAN BRUNO
Fellows, M. Fording, Comdr. (M.C.),
U. S. N. R., Lion No. 6 ALR
Smith, W. Jewell, Lt. Comdr. (M.C.), USNR
Lion 6, APBD, U. S. Navy ALR

SAN DIEGO
Bond, Floyd M.....625 Broadway, Z1 OP
Brown, Charles Wm.....Bank of America bldg., Z1 ALR
Brownell, Durwin Hall.....Medico-Dental bldg., Z1 ALR
Darrough, L. E., Comdr. (M.C.).....Naval Air Station ALR
Dennis, F. L.....2745 Barnson place, Z3 ALR
Durr, S. A.....Medico-Dental bldg., Z1 OP
Higbee, D. R.....3245 4th av., Z3 ALR
Hunt, W. R.....Lt. Comdr. (M.C.), Section Base, Z49 ALR
Merrill, H. G.....3245 4th av., Z3 OP
Mulsow, J. E., Lt. Comdr. (M.C.), 1818 6th av., Z1 OALR
Prendergast, John J.....2001 4th av., Z1 OP
Rees, Charles W., Lt. Comdr. (M.C.), 4496 Trias st., Z3 ALR
Ryan, Maurice J., Lt. (M.C.), USNR
4480 Hermosa Way, Z3 ALR

SAN FRANCISCO
Awtrey, H. H., Lt. Comdr. (M.C.),
U. S. Naval Hospital, Treasurer Island ALR
Barkan, Hans.....2351 Clay st., Z9 OP
Barkan, Otto.....490 Post, Z2 OP
Brownsberger, Sidney
Capt. (M.C.), Letterman General Hosp. OP
Cordes, Frederick C.....384 Post, Z8 OP
Davies, Windsor., U. S. Naval Hospital, Treasure Island OP
Edgerton, Ambrose E.....450 Sutter, Z8 OP
Fishman, Louis Z., Green's Eye Hosp.,
1801 Bush st., Z9 ALR
Fitzhugh, William McP., Jr.
Lt. Comdr. (M.C.), 384 Post, Z8 ALR
Gidoll, Sidney H.....516 Sutter, Z2 ALR
Hand, Frank.....450 Sutter, Z8 ALR
Hebert, Arthur W.....450 Sutter, Z8 OALR
Horner, Warren D., Capt. (M.C.), USNR,
2518 Jackson st. OP
Irvine, Robert Steele .....490 Post, Z2 OALR
Kobrak, Heinrick G.....Capt. (M.C.), Fort Mason ALR
Kress, George H., Apt. 506, 2200 Leavenworth st., Z11 OALR
Lachman, George S., Lt. Comdr. (M.C.),
2201 Broadway, Z15 OP
McCool, J. L.....450 Sutter, Z8 OP
Master, John R., Lt. (M.C.), Letterman General Hospital ALR
Monson, LaFayette.....450 Sutter, Z8 ALR
Nethercut, Ruth A.....450 Sutter, Z8 ALR
O'Connor, Roderic P.....450 Sutter, Z8 OP
Pierce, George W.....490 Post st., Z2 PI S
Pischel, D. K.....490 Post, Z2 OP
Pischel, Kasper.....490 Post, Z2 OP
Rawlins, Aubrey Gatliff.....384 Post, Z8 ALR
Rodin, Frank Highman.....490 Post, Z2 OP
Rome, Sol, Capt. (M.C.),
Station Dispensary, Fort Mason OP
Selfridge, G. L.....1000 California st., Z8 ALR

SAN JOSE
Jordan, Philip J.....910 Medico-Dental bldg., Z20 ALR
Martin, Paul T.....235 E. Santa Clara, Z20 ALR

CALIFORNIA — Continued

SAN LEANDRO
Lower, R. A.....Comdr. (M.C.), 670 Haas av. **ALR**

SAN MATEO
Mohun, Meade.....23 Second av. **ALR**

SAN PEDRO
Earl, Harry D.....479 Sixth **ALR**
Snow, Harold L.....639 W. 9th **OP**

SANTA ANA
Dixon, W. E.....1617 E. 4th **OALR**
Hutchinson, Ben B..Capt. (M.C.), Station Hosp., SAAAB **OP**
Maxwell, Harvey C...Lt. Col. (M.C.), 1718 N. Main st. **OALR**
Ogus, Alfred, Lt. (M.C.),
School of Aviation Medicine, S.A.A.A.B. **ALR**

SANTA BARBARA
Faier, Herman I., Maj. (M.C.), Hoff General Hospital **OP**
Hombach, Frank J.....1421 State **OP**
Jean, George W.....2774 Puesta del Sol Road **OP**
Johnston, William H.....1515 State **OALR**
Mellinger, William J.....323 E. Sola st. **ALR**

SANTA CRUZ
Ivins, H. M.....R.F.D. 4, Box 36 **OALR**

SANTA MARIA
Mesirow, Maurice E.....117 E. Cook **OP**

SANTA MONICA
Shellman, John L.....2731 Washington av. **OP**

SHOEMAKER
Cornelius, C. E., Lt. Comdr. (M.C.), U. S. Naval Hospital **ALR**
Hoxie, Derrick, Lt. Comdr. (M.C.),
U. S. Naval Receiving Barracks **ALR**

SOUTH PASADENA
Hall, Lawrence H.....1520 Oak st. **OALR**

STOCKTON
Sasiaw, Lewis Benjamin.....Bank of America bldg., Z5 **ALR**

STOCKTON FIELD
Birge, H. L.....Capt. (M.C.), A.F.A.T.S. **OP**

VALLEJO
Dexter, Milton A.....416 Georgia **ALR**
Madeley, Howard Randall.....727 Sonoma **OP**

VENTURA
Raines, Morris M.....34 N. Ash st. **ALR**

VISALIA
Keiper, Geo. F.....113 N. Church **ALR**
Sallander, F. W.....212 W. Main **OALR**

WEST LOS ANGELES
Guggenheim, Louis K.....1033 Gayley av., Z24 **ALR**
Hess, Wm. L.....1610 Sawtelle blvd. **OALR**

WHITTIER
Smith, H. A.....226 N. Greenleaf av. **OALR**

COLORADO

ALAMOSA
Herriman, L. L.....Physicians bldg. **OALR**

BOULDER
Fischer, V. B.....Mercantile Bank bldg. **OALR**
Spencer, Frank R.....Physicians bldg. **OALR**

BUCKLEY FIELD
Eimer, C. E., Maj. (M.C.), Medical & Psychology Unit **ALR**
Rudolph, Carl J., Maj. (M.C.), Eye Clinic Station Hosp. **OP**

COLORADO SPRINGS
Brobeck, von Haller.....Ferguson bldg. **OP**
Chapman, Katharine H.....304 Burns bldg. **OP**
Chapman, S. Jefferson.....Burns bldg. **ALR**
George, Arthur R..Capt. (M.C.), Hdqrs. Second Air Force **ALR**
Kettelkamp, F. O.....Ferguson bldg. **ALR**
Lamberson, William Henry....First National Bank bldg. **ALR**
Neeper, Edward R.....Exchange National Bank bldg. **OP**
Stine, George H.....Burns bldg. **OP**

DENVER
Bane, W. M.....1612 Tremont Place, Z2 **OP**
Baum, H. L.....Republic bldg., Z2 **ALR**
Beyer, Theodore E.....227 Sixteenth st., Z2 **ALR**
Carmody, T. E.....Metropolitan bldg., Z2 **ALR-PI S**
Collins, E. W.....1578 Humboldt, Z6 **ALR**
Cooper, Kemp G.....Metropolitan bldg., Z2 **ALR**
Crisp, William H.....Metropolitan bldg, Z2 **OP**
Danielson, Ralph W.....Metropolitan bldg., Z2 **OP**
Davis, Leo Lloyd.....1437 Monaco parkway, Z7 **OP**
Deems, Myers B., Maj. (M.C.),
Fitzsimmons General Hospital, Box 6087, Z8 **ALR**
Gale, M. Jean.....Republic bldg., Z2 **OP**
Greene, Laurence W.....Republic bldg., Z2 **ALR**
Gsell, George Franklin.....Capt. (M.C.), Lowry Field **OP**
Halsted, Fred S.....Metropolitan bldg., Z2 **OALR**
Hickey, Harold L.....Republic bldg., Z2 **ALR**
Laff, H. I.....Metropolitan bldg., Z2 **ALR**
Levy, Robert.....Metropolitan bldg., Z2 **ALR**
Leyda, James H.....Metropolitan bldg., Z2 **ALR**
Long, John C.....Metropolitan bldg., Z2 **OP**
McCaw, John A.....Majestic bldg., Z2 **OP**
McKeown, Elmer E.....Republic bldg., Z2 **OALR**
Marcove, Maurice E.....1612 Tremont pl., Z2 **OP**
Murphy, Rex L.....Metropolitan bldg., Z2 **ALR**
Ohmart, Walter A.....Republic bldg., Z2 **OP**
O'Rourke, D. H.....Republic bldg., Z2 **OP**
Pattee, George L.....612 Republic bldg., Z2 **ALR**
Reynolds, Edna M.....227 Sixteenth, Z2 **OP**
Sedwick, W. A.....Republic bldg., Z2 **OP**
Shields, J. M.....Metropolitan bldg., Z2 **OP**
Smith, Robert A., Lt. Col. (M.C.),
Box 6063, Fitzsimons General Hospital, Z8 **ALR**
Walker, C. E.....1732 High st., Z6 **OP**
Walker, Charles E., Jr., Maj. (M.C.), 1732 High st., Z6 **OP**
Youngerman, William M., Maj. (M.C.),
Station Hospital, Lowry Field **ALR**

GRAND JUNCTION
Cary, G. C.....Canon block **OALR**
Rigg, James P.....227 N. 5th **OALR**
Waldapfel, Richard.....De Merschman Gardens **ALR**

COLORADO — Continued

GREELEY
Atkinson, Thomas Ernest.....Coronado bldg. ALR
Lehan, James W.....1620 Twelfth av. OALR
Ringle, Charles A.....912 Ninth av. ALR

LA JUNTA
Farnsworth, M. A.....Opera House Apt. bldg. OALR

LONGMONT
Hageman, George Ricker.....Longmont Hospital ALR
Sidwell, Clarence Edward.....608 Fourth av. ALR

PUEBLO
Earnest, Clarence E.....Thatcher bldg. ALR
Hopkins, Guy H.....702 N. Main OP
Lassen, Fritz.....Pueblo Clinic OALR
Porter, Geo. L.....Capt. (M.C.), Army Air Base OP
Rusk, Harvey Schenck.....Colorado bldg. OALR

SALIDA
Thompson, Lester Emmit.....Woolworth bldg. ALR

STERLING
Elliff, Edgar Alonzo.....216 N. 3rd. ALR
Palmer, Frank E.....Box 267 OALR

TRINIDAD
Richie, L. T.....101 E. Main OALR

CONNECTICUT

BRIDGEPORT
Dewitt, Edward Nicholas.....881 Lafayette, Z4 OP
Garlick, George B.....144 Golden Hill, Z3 ALR
Kaplan, Leon.....881 Lafayette st., Z4 OP
Smith, Dorland.....881 Lafayette, Z4 OP A
Turchik, Frank.....1831 Barnum av., Z8 ALR

DANBURY
Sarnoff, Jack.....Capt. (M.C.), 16 Fifth av. ALR

GREENWICH
Miller, John.....63 North ALR
Yost, W. M.....Lake av. OP

HARTFORD
Connor, Joseph Joyce.....750 Main st., Z3 OP
Davis, James Edward.....179 Allyn, Z3 ALR
Hogan, W. L.....750 Main, Z3 OP
Katz, Dewey.....99 Pratt, Z3 OP
Lewis, Samuel D.....49 Pearl st., Z3 OP
Little, Milton F.....49 Pearl, Z3 OP
Snelling, P. W.....179 Allyn, Z3 ALR
Whalen, Edward J.....750 Main, Z3 ALR
Zeman, Michael S., Capt. (M.C.), 296 North Quaker Lane ALR

MIDDLETOWN
Chase, Carl C.....121 Main OP
Joyce, Wm. M.....121 Main st. ALR

NEW BRITAIN
Schechtman, Charles T.....81 W. Main ALR

NEW HAVEN
Berman, Harry L.....1142 Chapel, Z11 ALR
Canfield, Norton....Lt. Col. (M.C.), 789 Howard av., Z4 ALR
Ferguson, R. J.....59 College, Z10 OALR
Flynn, Charles T.....41 Trumbull, Z10 ALR
Latimer, Marvin L.....129 Whitney av. Z10 ALR
MacCready, Paul B.....442 Temple, Z11 ALR
Ryder, Wm. H.....291 Whitney av., Z11 OP
Sheahan, William L.....59 College, Z10 OALR
Silverberg, Samuel Joshua.....201 Park st., Z11 ALR
Smith, Norman N.....291 Whitney av., Z11 ALR
Sperry, Frederick N.....107 Whitney, Z10 ALR
Yudkin, Arthur Meyer.....257 Church, Z10 OP

NEW LONDON
Bacon, Lewis H., Lt. Comdr. (M.C.), U. S. Submarine Base Hospital OP
Stone, Leslie O., Capt. (M.C.), U. S. Naval Dispensary, Submarine Base ALR
Wilson, Frank E.....302 State ALR

RAWAYTON
Hurd, L. M.....Box F ALR

STAMFORD
Murray, Henry J.....53 South ALR
Turnley, William H.....One Atlantic ALR

TORRINGTON
Hill, E. S.....51 Main ALR

WATERBURY
Good, William Murray.....63 Center, Z18 ALR

WEST HARTFORD
Bausch, Carl P., Comdr. (M.C.), USNR, 37 Sulgrave rd., Z7 OP
Smith, E. Terry.....P. O. Box 42 OP

WILLIMANTIC
Arnold, Morton.....781 Main ALR

WINDSOR LOCKS
Vrooman, Carl O.....Maj. (M.C.), Bradley Field OP

DELAWARE

WILMINGTON
LaMotte, William O.....Medical Arts bldg., Z9 ALR
Mayerberg, Emil R.....Medical Arts bldg., Z9 ALR
Pierson, William M.....Medical Arts bldg., Z9 ALR
Strikol, A. J.....621 Delaware av., Z9 ALR

DISTRICT OF COLUMBIA

WASHINGTON
Alexander, Samuel A.....1830 Eye st. N. W., Z6 ALR
Andrews, A. H., Jr., Lt. (M.C.), Med. Research Sec. Bureau of Aeronautics, Navy Dept., Z25 ALR
Bell, John Gordon.....1835 Eye st. N. W., Z6 ALR
Bockoven, Sterling....1752 Massachusetts av. N. W., Z6 OP
Bradley, Jeter C.....900 Seventeenth st., N. W., Z6 ALR
Breeding, Earl G.....1801 Eye st., N. W., Z6 ALR
Brooks, Richmond C. A.....1150 Connecticut av., Z6 ALR
Clayton, Milton B.....Southern R. R. Office bldg., Z13 ALR
Constantinople, P. S.....1801 Eye st., N. W., Z6 ALR
Costenbader, Frank D.....1150 Connecticut av., Z6 OP
Cox, Ronald Atmore..1779 Massachusetts av., N. W., Z6 OP
Cummings, Edward Joseph.....1835 Eye st. N. W., Z6 ALR
Davies, H. F.....1835 Eye st. N. W., Z6 ALR

WASHINGTON, D. C. — Continued

Davis, David.....1835 Eye st. N. W., Z6 ALR
Davis, William Thornwall 927 Seventeenth st. N. W., Z6, OP
Delaney, Adrian J., Comdr. (M.C.), USN,
1519 31st st. N.W. ALR
Dessoff, Joseph.....1726 Eye st. N. W., Z6 OP
Downey, Harold R.
Lt. Comdr. (M.C.), 1740 M st. N. W., Z6 OP
Enlows, Ella M. A.....1816 R st. N. W., Z9 ALR
Fischer, Aubrey.....1835 Eye st. N. W., Z6 ALR
Flynn, James A.....1511 Rhode Island av. N. W., Z5 ALR
Gill, G. Bache.....1726 Eye st. N. W., Z6 ALR
Goodman, E. Leonard.....1801 Eye st. N. W., Z6 OP
Henning, Carl.....1752 Massachusetts av. N. W., Z6 OP
Hogsett, Smith Fuller, Jr., Lt. (M.C.), U.S.N.R.
3009 Erie st. S. E., Apt. B-263, Z20 OP
House, Hugh O.....1835 Eye st. N. W., Z6 ALR
Johnson, Don.....1835 Eye st. N. W., Z6 ALR
Kearny, Richard.....2311 Connecticut av., N. W., Z8 ALR
Lamb, Robert Scott.....Stoneleigh court, Z6 OP
Luber, Samuel.....1801 Eye st. N. W., Z6 ALR
Lynch, Mercer G., Major (M.C.), Quarters 113,
New Section, Walter Reed Hospital ALR
Meloy, William C.
Lt. Comdr. (M.C.), 2019 R. st. N. W., Z9 ALR-PI S
Moffett, Daniel B.....1150 Connecticut av. N. W., Z6 ALR
Morgan, William A.....1835 Eye st. N. W., Z6 ALR
Morrison, William W., Comdr. (M.C.),
Naval Dispensary, Naval Department, Z25 ALR
Moss, L. Conner
Comdr. (M.C.), 2223 Observatory pl. N. W., Z7 OP
Novick, Joel N.....1726 Eye st. N. W., Z6 ALR
Pendexter, R. Stevens.....Farragut Medical bldg., Z6 OP
Posner, Adolph, Capt. (M.C.),
General Dispensary, 2D201 Pentagon bldg., Z25 OP
Riwchun, M. H...Maj. (M.C.), Walter Reed Hospital, Z1 OP
Rones, Benjamin.....1610 Eye st. N. W., Z6 OP
Runk, R. L.
Washington Sanitarium and Hospital, Tacoma Park, Z12 ALR
Sawyer, Leroy Lee, Jr.....Farragut Medical bldg., Z6 ALR
Shepherd, Elmer Roberts.....1746 K st. N. W., Z6 ALR
Sheppard, E. A. W.....927 Seventeenth st. N. W., Z6 OP
Tegge, Charles William.....1801 Eye st. N. W., Z6 ALR
Trible, G. B.....1150 Connecticut av. N. W., Z6 OALR
Vann, Homer King.....410 Cedar st. N. W., Z12 ALR
Wilber, Inez Edith.....1746 K st. N. W., Z6 OP
Wilkinson, Oscar.....1408 L st. N. W., Z5 OALR
Wilkinson, Richard W.....1408 L st. N. W., Z5 ALR
Winer, Albert S.....1835 Eye st. N.W., Z6 ALR
Zinkhan, A. M.....1801 Eye st. N. W., Z6 ALR

FLORIDA

AVON PARK

Ricchiuti, A. George, Capt. (M.C.),
Station Hospital, Avon Pk. Bomb Range ALR

CAMP BLANDING

Goldsmith, Jacob.....Maj. (M.C.), Station Hospital OP

CLEARWATER

Nickle, Millen Alexander.....Box 305 ALR
Thygeson, Phillips.....Maj. (M.C.), 971 Eldorado OP

COCOANUT GROVE

Hutchinson, Abbott T.....4086 Roynolds av. ALR

CORAL GABLES

Cracovaner, Arthur J.
Major (M.C.), AAF Regional Station Hospital ALR
Hasty, F. E.....1203 Asturia ALR
Unsworth, Arthur C., Lt. Col. (M.C.),
709 N. Greenway Drive, Z34 OP

EGLIN FIELD

Harlowe, Harold Douglas.....Capt. (M.C.), Sta. Hosp. ALR

FORT LAUDERDALE

Connor, Arthur B.....Sweet bldg. ALR

FORT MYERS

Hearn, Paul P., Capt. (M.C.),
Station Hospital, Buckingham Field ALR
Merrick, Charles Gordon.....Leon bldg. ALR

JACKSONVILLE

Knauer, William J.....Buckman bldg., Z2 OALR
O'Dell, John C., Jr.....Medical Arts bldg., Z4 ALR
Richardson, Shaler.....111 W. Adams, Z2 OP
Taylor, H. Marshall.....111 W. Adams, Z2 ALR
Wilson, Alpheus K.....334 St. James bldg., Z2 ALR

KEY WEST

Wattles, F. Merrill.Lt. Comdr. (M.C.), U. S. Naval Hosp. ALR

MERRITT ISLAND

Thomsen-von Colditz, G.....Dun Huntin OALR

MIAMI

Black, Nelson Miles.....Huntington bldg., Z32 OP
Brown, Andrew George.....1010 Huntington bldg., Z32 OP
Chandler, G. E.....Huntington bldg., Z32 ALR
Crossley, E. R.....6821 N. W. 4th av., Z38 OALR
deBoe, M. P.....Huntington bldg., Z32 OALR
Dunaway, Carl E.....Huntington bldg., Z32 ALR
Hodsdon, B. F.....Box 923, Z6 OALR
Ingersoll, J. M.....1700 S. Bayshore lane, Z33 ALR
Mendel, James H.....Ingraham bldg., Z32 ALR
Palmer, Bascom H.....Huntington bldg., Z32 OALR
Park, M. B.....401 Ingraham bldg., Z32 OP
Weinstein, Philip.....Ingraham bldg., Z32 ALR

MIAMI BEACH

Dart, M. O., Major (M.C.),
AAF Redistribution Station No. 2 ALR
Denman, Francis E.....541 Lincoln road, Z39 ALR
Hotchkiss, W. T.....541 Lincoln road, Z39 ALR
Repass, R. E.....605 Lincoln road ALR
Richardson, John Roberts
Lt. Comdr (M.C.), U.S.N.R., 29C-Venetian Way, Z39 ALR
Theodore, F. H.....Capt. (M.C.), 1210 Lenox av., Z39 OP

ORLANDO

Campbell, Mac D., Capt. (M.C.),
Station Hospital, A.A.F.S.A.T. OALR
Ingram, Hellis Carlyle.....Capt. (M.C.), 842 Laurel OP
Ingram, L. C.....842 Laurel OALR
Johnston, Hewitt.....303 Exchange bldg. ALR
Lockwood, Chester C., Capt. (M.C.),
Station Hospital, A.A.F.T.A.C. OP
Shoemaker, Samuel A.....613 S. Hyer st. OALR

PALM BEACH

Drooker, J. C., Major (M.C.),
Ream General Hospital, Box 9 ALR

FLORIDA — Continued

PENSACOLA

Borley, William E., Lt. Comdr. (M.C.), U.S.N.R.
School of Aviation Medicine, Naval Air Station **OP**
Cook, Raymond C.
Lt. Comdr. (M.C.), U.S.N.A.S. Dispensary **OP**
Gilbert, Joseph G., Lt. Comdr. (M.C.),
Naval Hospital, Naval Air Training Center **ALR**
Heinberg, Charles J......Medical Center **ALR**
Lischkoff, M. A......Blount bldg. **OALR**
McLane, Jesse Newman.....Blount bldg. **ALR**
Riggs, Ralph Hearsey
Lt. Comdr. (M.C.), Naval Air Station **ALR**
Wolpaw, B. J., Lt. Comdr. (M.C.),
Naval Air Station Dispensary **OP**
Wright, Edward S., Lt. Comdr. (M.C.), U.S.N.R.
Naval Hospital, Naval Air Training Center **ALR**

RICHMOND

Levin, Nathaniel M.
Comdr. (M.C.), U. S. Naval Air Station **ALR**

ST AUGUSTINE

Grace, Charles C......145 King st. **ALR**

ST. PETERSBURG

Heard, Mary K......(Retired) R.F.D. 2, Box 75, Z6 **OP**
McNay, Miller Ost.....472 Central av., Z5 **ALR**
Nelson, Orville N......1035 39th av., Z6 **OALR**
Post, W. L., Maj. (M.C.),
102 52nd st., Bennett Beach, Z7 **OALR**
Solomon, Henry D......Power and Light bldg., Z5 **ALR**

SANFORD

Williams, R. J.
Lt. Comdr. (M.C.), Naval Air Station Dispensary **ALR**

SEBRING

Manigan, Thomas P......Maj. (M.C.), Hendricks Field **ALR**

TALLAHASSEE

McLemore, Carl Sloan, Capt. (M.C.),
Station Hospital, Dale Mabry Field **ALR**

TAMPA

Clarke, Clement C., Major (M.C.),
Station Hospital, MacDill Field, Z8 **OP**
Duke, R. R......706 Franklin, Z2 **ALR**
Forbes, Sherman B......706 Franklin, Z2 **OALR**
Lowry, B. W......Citizens Bank bldg., Z2 **OALR**
Taylor, Joseph W......706 Franklin, Z2 **OALR**

WEST PALM BEACH

Miller, Alice R......505 Guaranty bldg. **OALR**
Sayad, William Y......Harvey bldg. **OALR**

GEORGIA

ALBANY

Callahan, Alston.....Lt. (M.C.), R.F.D. 4, Box 26X **OP**

ATLANTA

Baird, James M......Medical bldg., Z3 **OP**
Brawner, Leon Edward.....Medical Arts bldg., Z3 **ALR**
Brown, Lester A......478 Peachtree st. N.E., Z3 **ALR**
Burgess, Taylor S., Lt. Comdr. (M.C.),
Medical Arts bldg., Z3 **ALR**
Calhoun, F. Phinizy.....478 Peachtree st. N. E., Z3 **OP**
Campbell, W. E., Jr......Medical Arts bldg., Z3 **OP**
Clay, G. E......Medical Arts bldg., Z3 **OP**
Dutrow, H. V.
Lt. Col. (M.C.), Lawson General Hospital **OALR**
Equen, M. S......144 Ponce de Leon av. N. E. **ALR**
Hallum, Alton V......478 Peachtree st. N. E., Z3 **OP**
Howell, Stacy C......478 Peachtree st. N. E., Z3 **OP**
Lentine, Joseph...Capt. (M.C.), Lawson General Hospital **ALR**
Lokey, H. M......Medical Arts bldg., Z3 **OALR**
McDougall, J. Calhoun.....Medical Arts bldg., Z3 **ALR**
McDougall, William L......Doctors' bldg., Z3 **ALR**
Martin, William O., Jr......478 Peachtree st. N.E., Z3 **OP**
Veirs, Everett R....Maj. (M.C.), Lawson General Hospital, **ALR**
Vorisek, E. A....Maj. (M.C.), 8 E. Shadowlawn av. N. E., **OP**
Warren, William C., Jr......Doctors' bldg., Z3 **ALR**

AUGUSTA

Budetti, Joseph A.
Capt. (M.C.), Oliver General Hospital **ALR**
Diess, Robert G., Maj. (M.C.),
Oliver General Hospital, Forest Hills **OP**
Lewis, Samuel J......S. F. C. bldg. **ALR**
Shearer, Geo. M.
Lt. Col. (M.C.), Station Hospital, Camp Gordon **ALR**

CAMP STEWART

Brandfield, I. I......Capt. (M.C.), Station Hosp. **ALR**

MACON

Seip, Walter R...Capt. (M.C.), Sta. Hosp., Camp Wheeler **OP**
Smith, J. Allen.....Doctors' bldg. **ALR**
Sporn, Hyman, Maj. (M.C.),
Station Hospital, Camp Wheeler **ALR**

ROBINS FIELD

Johnson, Gayle T......Maj. (M.C.), Station Hospital **ALR**

ROME

Coleman, Benjamin, Capt. (M.C.), Battey General Hosp. **ALR**
Smith, George B......Harbin Hospital **OALR**

SAVANNAH

Collins, Braswell E., Maj. (M.C.),
Sta. Hosp., Hunter Field **ALR**
Faggert, George H......18 W. Oglethorpe av. **ALR**
Foote, William D., Capt. (M.C.),
Station Hospital, Hunter Field **OP**
Good Palmer W......Capt. (M.C.), Hunter Field **OALR**
Lang, George Hermanne.....202 E. Liberty **ALR**
McCollom, Robert L., Major (M.C.),
397th Bomb Group, Hunter Field **ALR**

THOMASVILLE

Gaines, T. R., Maj. (M.C.), Finney General Hospital **OP**
Neidlinger, William J., (M.C.), Finney General Hospital **ALR**

HAWAII

HILO

Crawford, H. E......139 Kinoole st. **OP**

HONOLULU

Moffat, Harold F.
The Clinic, South Hotel st. at Thomas Square **OP**
Pinkerton, Forrest Joy.....Young Hotel bldg., Z9 **OALR**
Trexler, Clarence W., Lt. Comdr. (M.C.), 50 Dowsett av. **ALR**
Van Poole, G. McD......Young bldg., Z9 **OALR**

IDAHO

NAMPA

Shawhan, Glenn E.....Dewey Scales bldg. OALR

POCATELLO

Clothier, Joseph.....Kane bldg. OALR
Clothier, William L.....407-11 Kane bldg. OP
Pond, Casper W.....Carlson bldg. ALR

ILLINOIS

ALTON

Jones, F. W.....101 E. Broadway OALR
Middleton, Harry E.....206A W. 3rd OALR
Rawlins, Grove A.....206A W. 3rd ALR

AURORA

Darmer, George A.....Terminal bldg. OALR

BELLEVILLE

Otrich, Grover C.....Commercial bldg. ALR
Trowbridge, Bernard C.....Lt. (M.C.), Scott Field ALR
Schwartz, F. O.....230 E. Main OP

BLOOMINGTON

Elliott, J. Norman.....Griesheim bldg. ALR
Gailey, Watson W.....1008 N. Main OALR
Hartenbower, George E.....Unity bldg. ALR
Watkins, Harold R.....Griesheim bldg. ALR

CAMP ELLIS

Allen, Wilmer L.....Major (M.C.), Station Hospital ALR
Hurwitz, Paul.....Maj. (M.C.), Station Hospital OP
Smiley, Wm. A., Maj. (M.C.).....66 Field Hospital ALR

CENTRALIA

Hirschfelder, Max Kurt.....408 W. 2nd OP

CHAMPAIGN

Albers, Ed. C.....104 W. Clark OP
Ford, Hanby Lewis.....Robeson bldg. OALR

CHANUTE FIELD

Stoner, Cyrus H.....Major (M.C.), Box 523 OP

CHICAGO

Ackerman, Walter G.....122 South Michigan av., Z3 OP
Allen, Thomas Dyer.....122 South Michigan av., Z3 OP
Andrews, A. H.....25 East Washington, Z2 OALR
Apple, Carl.....55 East Washington, Z2 OP
Barnett, Irving F.....30 North Michigan av., Z2 OALR
Becker, Oscar J.....310 South Michigan av., Z4 ALR
Beem, Ione F.....30 North Michigan av., Sec. 2, Z2 OALR
Bellows, John G., Maj. (M.C.),
c/o Louis Bellows, 928 Ainslie st. OP
Bernheimer, L. Benno.....104 S. Michigan av., Z3 ALR
Blonder, Edwin J.....185 N. Wabash av., Z1 ALR
Blumkoltz, Bruno.....185 N. Wabash av., Z1 ALR
Bothman, Louis.....310 S. Michigan av., Z4 OP
Brandenburg, Nora B.....720 N. Michigan av., Z11 ALR
Brawley, F. E.....30 N. Michigan av., Z2 OP
Bredlau, Ernest A.....2753 W. North ALR
Brooks, Clyde Montgomery, Capt. (M.C.),
Gardiner General Hospital, Z15 ALR
Brooks, Jack E.....712 Grace st., Z13 ALR
Brunner, Hans.....307 North Michigan av., Z1 ALR
Carelli, Paul Vincent.....3626 W. Chicago av., Z51 OP
Chainski, Edward L.....130 N. Wells, Z6 ALR
Christoph, Carl H.....30 N. Michigan av., Z2 OALR
Cleff, Oscar.....55 E. Washington, Z2 OALR
Clement, C. C.....30 N. Michigan av., Z2 OALR
Comee, W. C.....6320 N. Kenmore av., Z20 OALR
Coombs, A. J.....30 N. Michigan av., Z2 ALR
Cottle, Maurice H.....30 N. Michigan av., Z2 ALR
Crage, Francis M.....25 E. Washington st., Z2 OP
Cushman, Beulah.....25 E. Washington, Z2 OP
Davidson, H. P.....30 N. Michigan av., Z2 OP
Delph, John F.....700 N. Michigan av., Z11 ALR
Dennis, George J.....25 E. Washington, Z2 ALR
Dougherty, Clifford L.....55 E. Washington, Z2 ALR
Edelman, Maurice I.....Lt. (M.C.), 849 Gunnison st. ALR
Fabricant, Noah D.....185 N. Wabash av., Z1 ALR
Findlay, E. K.....30 N. Michigan av., Z2 OP
Fisher, W. A.....156 N. Franklin st., Z6 OALR
Fitzgerald, J. P.....30 N. Michigan av., Z2 OP
Fitzgerald, J. R.....30 N. Michigan av., Z2 OP
Fowler, Earle B.....55 E. Washington, Z2 OP
Fox, Noah.....841 E. 63rd, Z37 ALR
Frank, Ira.....104 S. Michigan av., Z3 ALR
Friedberg, Stanton A.
Capt (M.C.), 455 Barry av., Lincoln Park Station, Z14 ALR
Friedman Elmer Arthur.....6733 Newgard av., Z26 ALR
Gamble, Richard Cotter.....30 N. Michigan av., Z2 OP
Glatt, Morris A.....55 E. Washington, Z2 ALR
Goldenburg, Michael.....104 S. Michigan av., Z3 OP
Gradle, H. S.....58 E. Washington, Z2 OP
Green, H. S., Lt. Comdr (M.C.),
USNR, 6200 N. Kenmore av., Z40 OALR
Greenwood, Glenn J.....55 E. Washington, Z2 ALR
Guibor, George P.....30 N. Michigan av., Z2 OP
Gustafson, Olga Frideberg.....185 N. Wabash av., Z1 OP
Guttman, M. Reese.....30 N. Michigan av., Z2 ALR
Hagens, Elmer William.....30 N. Michigan, Z2 ALR
Hall, Alice K.....25 E. Washington, Z2 ALR
Hayden, Jerome William.....4458 Madison st., Z24 ALR
Hill, Howard E., Maj. (M.C.), Station Hosp.,
A.A.F.T.S., 1660 E. Hyde Park blvd., Z15 ALR
Hipskind, Myron Maxwell.....6060 Drexel av., Z37 ALR
Hoeltgen, Maurice M.....1607 West 51st st., Z9 OP
Hoffman, L. G.....30 N. Michigan av., Z2 OALR
Hoffman, William A.....30 N. Michigan, Sec. 2., Z2 OP
Holinger, Paul H.....700 N. Michigan av., Z11 L
Hollender, Abraham R.....307 N. Michigan av., Z1 ALR
Holt, Helen.....720 North Michigan av., Z11 OP
Howard, William H.....9152 Commercial av., Z17 OP
Jacobs, Milton.....122 S. Michigan av., Z3 OP
Jordon, George T.....30 N. Michigan av., Z2 OALR
Judd, Delbert K.....950 E. 59th, Z37 ALR
Kammerling, Theodore S.....616 N. Central av., Z44 OALR
Kaufman, Samuel I.....185 N. Wabash av., Z1 OP
Kerwin, Raymond W.....310 S. Michigan av., Z4 OALR
Klien, Bertha A.....58 E. Washington, Sec. 2., Z2 OP
Koch, Harold J.....6235 S. Kedzie av., Z29 OP
Kraft, Oscar H.....1518 N. Dearborn Parkway, Z10 OP
Krause, Arlington C.....950 E. 59th, Z37 OP
Kronfeld, Peter C.....904 W. Adams, Z7 OP
Kulvin, Max M.....55 E. Washington, Z2 OALR
LaReau, Hector G.....4803 Lincoln av., Z25 ALR
Lazar, Nathan K.....434 Roscoe st., Z13 OP
Lebensohn, James E.
Comdr. (M.C.), 20 E. Delaware place, Z11 OALR
Lederer, Francis L., Comdr. (M.C.),
307 North Michigan av., Z1 ALR
Leech, Vernon M.....55 E. Washington, Z2 OP

ILLINOIS — Continued

Leshin, Norman.....5480 Cornell av., Z15 ALR
Lewy, Alfred.....25 E. Washington st., Z2 ALR
Lifschutz, Jacob.....310 S. Michigan av., Z4 OALR
Lindsay, John R.....950 E. 59th, Z37 ALR
Livingston, George S.....185 N. Wabash av., Z1 ALR
Lyons, H. R.....6 N. Michigan av., Z2 ALR
McBean, George M.....25 E. Washington, Z2 OALR
McBride, Linn Frederick.....122 S. Michigan av., Z3 ALR
McIntyre, George Fillmore.....25 E. Washington, Z2 ALR
McRoberts, Earl S.....55 E. Washington, Z2 ALR
Mann, William A.....30 N. Michigan av., Z2 OP
Meyer, Samuel J.....58 E. Washington, Z2 OP
Moncreiff, William F.....58 E. Washington, Z2 OP
Morwitz, Samuel M.....55 E. Washington, Z2 ALR
Mundt, G. Henry.....30 N. Michigan av., Z2 OALR
Muskat, Irving.....25 E. Washington, Z2 OALR
Nance, Willis O.....6845 Euclid av., Z49 OALR
Nash, Herbert T.....31 N. State, Z2 ALR
Nethercut, Glenway W.....25 E. Washington, Z2 OP
Noonan, W. J.....4753 Broadway, Z40 OALR
Novak, Frank J., Jr.....30 N. Michigan av., Z2 ALR
Nugent, Oscar B.....231 W. Washington, Z6 OP
O'Connor, Philip D.....30 N. Michigan av., Z2 OP
O'Connor, Thomas Philip.....30 N. Michigan av., Z2 ALR
Olkon, Dora Berkman.....58 E. Washington, Z2 OP
Orcutt, D. C.....55 E. Washington, Z2 OALR
Pearlman, S. J.....55 E. Washington, Z2 ALR
Pellettiere, Edmund V.....1530 N. Damen av., Z22 ALR
Perlman, Henry B.....950 E. 59th, Z37 ALR
Pollack, Samuel..Capt. (M.C.), 2909 W. Estes av., Z45 ALR
Puntenney, Irving.....720 N. Michigan av., Z11 OP
Roling, Edward A.....2753 W. North av., Z47 OP
Ross, Percy J.....104 S. Michigan av., Z3 ALR
Salinger, Samuel.....25 E. Washington, Z2 ALR
Savitt, Louis.....3403 Lawrence av., Z25 ALR
Scheffler, Milton Morris.....58 E. Washington st., Z2 OP
Scheribel, Karl J.....55 E. Washington, Z2 OP
Sciaretta, S. A.....55 E. Washington, Z2 ALR
Selinger, Elias.....104 S. Michigan av., Z3 OALR
Shambaugh, George E., Jr.....122 S. Michigan av., Z3 ALR
Shapira, Theodore M.....58 E. Washington, Z2 OP
Shapiro, S. L.....185 N. Wabash av., Z1 ALR
Smith, Hiram J.....30 N. Michigan av., Z2 OP
Snitman, Maurice F.....55 E. Washington st., Z2 ALR
Soboroff, Simon H.....1608 Milwaukee av., Z47 ALR
Sowers, Alva Boyd.....6 N. Michigan av., Sec. 2, Z2 OP
Steffenson, Olaf.....25 E. Washington, Z2 ALR
Strauss, J. F.....104 S. Michigan av., Z3 ALR
Sullivan, Clifford Patrick, Capt. (M.C.), 304 W. 63rd, Z21 OP
Theobald, W. H.....307 N. Michigan av., Z1 ALR
Tindal, John M.....5517 W. Fulton, Z44 OALR
Tivnen, Richard J.....30 N. Michigan av., Z2 OALR
Van Alyea, O. E.....135 S. La Salle, Z3 ALR
Vermeren, John C. Lt. Comdr. (M.C.), 4753 Broadway, Z40 ALR
Von der Heydt, Robert.....25 E. Washington, Z2 OP
Wescott, Virgil.....30 N. Michigan av., Z2 OP
Wojniak, Frank.....4649 S. Ashland av., Z9 ALR
Zekman, Theodore N...Lt. (M.C.), 1627 Hyde Park blvd. OP

CHICAGO HEIGHTS

Gorrell, T. J. H.....16th and Vincennes av. OALR

DANVILLE

Baldwin, H. E.....139 N. Vermillion OALR
Baldwin, Stephen Glidden.....Adams bldg. ALR
Fink, Otto E.....9 W. Madison OP

DECATUR

Hubble, William Fields.....Citizens bldg., Z30 ALR

DIXON

McNichols, W. A.....101 W. First, Z3 OALR

EAST ST. LOUIS

Hanson, William Ludwig.....Spivey bldg. ALR
Spitze, E. C.....Murphy bldg. OP

ELGIN

Russell, Roland D.....Room 808 Professional bldg. ALR

EVANSTON

Dodd, Oscar.....636 Church st. OP
Droegemueller, Wm. H., Maj. (M.C.), 116 Burnham pl. OP
Eubank-Jones, Miriam D.....944 Maple av. OP
Galloway, T. C.....636 Church st. ALR
Lawson, L. J.....636 Church st. ALR
McGarry, H. Isabelle.....636 Church st. OP
Soper, Gail Raney.....636 Church st. OP
Williams, Thomas J.....1604 Chicago av. ALR

FORT SHERIDAN

Jerome, Bourne.....Maj. (M.C.), Station Hospital OP

FREEPORT

Clark, J. Sheldon.....509 State Bank bldg. OALR

GREAT LAKES

Brindley, Benjamin, Comdr. (M.C.), U. S. Naval Hosp. OP
DeLozier, Leonard C., Lt. (M.C.), U. S. Naval Training Station ALR
Hornbogen, D. P. Lt. Comdr. (M.C.), U. S. Naval Hospital OP
Koebbe, E. E.....Comdr. (M.C.), U. S. Naval Hospital ALR
Potter, Jacob J., Lt. Comdr. (M.C.), Great Lakes Naval Hospital ALR
Row, D. Hamilton, Lt. Comdr. (M.C.), Dispensary No. 109, U.S.N.T. Station OP
Sage, Harry M., Comdr. (M.C.), McIntire Disp. Hospital, G.L.N.T.S. OP

HINSDALE

Hara, H. James.....Hinsdale Sanitarium and Hospital ALR

HOMEWOOD

Dillon, Elmo E.....1952 Ridge road OALR

JACKSONVILLE

Adams, A. L.....Professional bldg. OALR

JOLIET

Lennon, Robert Watson.....58 N. Chicago st. OP
Woodruff, George H.....250 N. Ottawa ALR
Woodruff, H. W.....250 N. Ottawa OALR

KANKAKEE

Geiger, C. W.....Arcade bldg. OALR

LA GRANGE

Ehrmann, Evelina W.....327 S. La Grange road OP

ILLINOIS — Continued

LAKE FORREST
Ashley, Byron J.....766 North Sheridan ALR

LA SALLE
Woods, R. H.....153 Marquette OP

MACOMB
Duntley, G. S.....Box 47 OALR

MATTOON
Jones, Florentine B.....416 South 17th ALR
Voigt, C. B.....1702 Broadway OALR

MAYWOOD
Spiesman, Irwin O.....1908 St. Charles rd. ALR

MOLINE
Flatley, T. J.....1518 Fifth av. OALR

OAK PARK
Palmer, Mahlon Phelps.....715 Lake ALR
Theobald, Georgiana Dvorak.....715 Lake ALR
Theobald, John J.....715 Lake ALR

OLNEY
Fisher, H. Noland.....106 N. Sliver st. OP

PARK RIDGE
Riser, Roy O.....120 Main OP

PEKIN
Glasford, S. T.....416 Elizabeth OALR

PEORIA
Blickenstaff, A. J.....Alliance Life bldg., Z2 OALR
Howard, H. Sargent.....418 Jefferson bldg., Z2 ALR
Owen, Walter E.....1242 Jefferson bldg., Z2 ALR
Williams, W. C.....Jefferson bldg., Z2 OALR

PONTIAC
Middleton, A. B.....OALR

QUINCY
Gabriel, C. K.....Illinois State Bank bldg. OALR
Stevenson, Walter.....W. C. U. bldg. OALR

RANTOUL
Straus, G. D.....Capt. (M.C.), Chanute Field ALR

ROCKFORD
Blomberg, T. E.....501 Seventh ALR
Fringer, W. R.....109 Sheridan st. OP

SCOTT FIELD
Allen, James H.....Maj. (M.C.), Station Hospital OP

SKOKIE
Wallner, Linden J...Maj. (M.C.), 4350 Bobolink Terr. ALR

SPRINGFIELD
Broadwell, Stuart, Jr.....103½ N. 5th ALR
Deal, John Francis.....522 E. Capitol av. OALR
Kerst, John Arthur.....Capt. (M.C.), R. R. No. 4 OP

STERLING
Brodrick, Frank W.....302 First av. OALR

STREATOR
Lester, Harry S.....Murray bldg. OALR

TAYLORVILLE
Duncan, Perry E.....101½ E. Main. OP

URBANA
Thacker, Edgar A.....602 W. University av. ALR

WINNETKA
Ballenger, Howard C.....723 Elm ALR

INDIANA

BLOOMINGTON
Geiger, Dillon.....Major (M.C.), N. Fee Lane ALR

BLUFFTON
Smith, H. Brooks.....303 S. Main OP

COLUMBIA CITY
Dyar, Edward William.....Lt. (M.C.) OP

CRAWFORDSVILLE
Lingeman, B. N.....Ben Hur bldg. ALR

EVANSVILLE
Ravdin, B. D.....Hulman bldg., Z18 OALR
Ravdin, M.....1666 Bayard Pk. dr., Z14 OALR
Stanton, Harmon La M.....Hulman bldg., Z18 OALR
Wesson, Thomas W.....124 S. E. 1st, Z8 ALR

FORT BENJAMIN HARRISON
Struble, Gilbert Carl
Lt. Col. (M.C.), Billings General Hospital OP

FORT WAYNE
Bulson, Eugene Loring.....347 W. Berry, Z2 OALR
Gessler, William F.....2902 Fairfield av., Z6 OALR

GARY
Carmody, Raymond Francis.....504 Broadway OP

HAMMOND
Kuhn, Hedwig S.....112 Rimbach OP
Kuhn, Hugh A.....112 Rimbach OALR
Lloyd, A. W.....Lloyd bldg. OALR
Shanedling, P. D.....5231 Hohman av. OP

HARTFORD CITY
Owsley, Guy A.....Col. (M.C.), 214 N. High ALR
Owsley, Robert W.....Major (M.C.), 214 N. High st. ALR

INDIANAPOLIS
Adams, D. S.....520 Hume-Mansur bldg., Z4 OALR
Alvis, Edmond Ochs.....320 Hume-Mansur bldg., Z4 OP
Bartley, Donald A.....706 Hume-Mansur bldg., Z4 OP
Berry, D. F.....Bankers Trust bldg., Z4 ALR
Brown, David Edward.....520 Hume-Mansur bldg., Z4 ALR
Clark, Cecil P.....922 Hume-Mansur bldg., Z4 OP
Craft, Kenneth L.....1002 Hume-Mansur bldg., Z4 ALR
Ellis, Bert E.....303 Hume-Mansur bldg., Z4 ALR
Garrett, John D.....Bar Association bldg., Z4 ALR
Harding, Myron S.....308 Hume-Mansur bldg., Z4 OP
Harris, Carl B.....319 Hume-Mansur bldg., Z4 OP
Hughes, W. F.....401 Hume-Mansur bldg., Z4 OP
Jones, David E.....Chamber of Commerce, Z4 ALR

INDIANA — Continued

Larkin, Bernard J.....305 Hume-Mansur, Z4 OP
Leasure, J. Kent.....611 Hume-Mansur bldg., Z4 ALR
Lingeman, Edward Laurel....315 Hume-Mansur bldg., Z4 ALR
Littell, J. Jerome .....603 Hume-Mansur bldg., Z4 ALR
McCaskey, C. H.....Guaranty bldg., Z4 ALR
Manion, Marlow W.....601 Hume-Mansur bldg., Z4 ALR
Mann, Mortimer.....323 Hume-Mansur bldg., Z4 OP
Masters, Robert J.....805 Hume-Mansur bldg., Z4 OP
Molt, William F.....Bankers Trust bldg., Z4 ALR
Norris, Mary Alice.....404 Hume-Mansur bldg., Z4 OP
Overman, F. V.....809 Hume-Mansur bldg., Z4 ALR
Pfafflin, C. A.....Bankers Trust bldg., Z4 OALR
Rubin, Milton R.....Capt. (M.C.), 3450 Winthrop, Z5 ALR
Rutherford, C. W.....501 Hume-Mansur bldg., Z4 OP
Sage, Russell A.....505 Hume-Mansur bldg., Z4 ALR
Seaton, Guy W.....424 Hume-Mansur bldg., Z4 ALR
Sparks, Alan Leo....Maj. (M.C.), 4310 Central av., Z5 ALR
Sputh, Carl B.....224 N. Meridian st., No. 301, Z4 ALR
Stevens, Ralph W.
Capt. (M.C.), Station Hospital, Stout Field, Z6 OP
Sudranski, Herbert F.....522 Hume-Mansur bldg., Z4 OP
Swan, John R.....915 Hume-Mansur bldg., Z4 ALR
Van Osdol, Harry A.....Chamber of Commerce bldg., Z4 ALR
Whitaker, Joel.....807 Hume-Mansur bldg., Z4 OALR
Wright, J. William.....301 Hume-Mansur bldg., Z4 ALR

KOKOMO

Adams, Charles Joseph.....Armstrong-Landon bldg. OALR

LAFAYETTE

Peterson, Joel A.....609 Lafayette Life bldg. ALR
Van Burskirk, Edmund Linford.....308-312 N. 8th OP

LOGANSPORT

Egan, Burton W.....408 North st. OALR

MARION

Braunlin, William H.....Marion National Bank bldg. OALR

MICHIGAN CITY

Krieger, G. M.....701 Washington OALR

MUNCIE

Deutsch, William, Jr..Major (M.C.), 1001 W. Gilbert st. ALR

RICHMOND

Holland, Emory E.....51 S. 8th OP

SOUTH BEND

Cassady, J. Vernal.....Sherland bldg., Z9 OALR
Rigley, Edward L.....107 E. Jefferson blvd., Z9 ALR
Savery, C. E.....Sherland bldg., Z9 OALR

TERRE HAUTE

Allen, Orris T.....Rose Dispensary bldg. OALR
Gillum, J. R.....221 S. 6th OALR
McBride, Noel S.....12 S. 7th OP
Stewart, Walter E.....Tribune bldg. OALR

UNION CITY

Ruby, F. McK.....235 W. Pearl OALR

IOWA

BURLINGTON

La Force, E. F.....Iowa State Bank bldg. OALR
Lee, Wayne Robert.....613 Medical Arts bldg. ALR
Pearson, George J.....320 N. 3rd ALR

CAMP DODGE

Flaherty, Norman W.....Capt. (M.C.) OP

CARROLL

McConkie, Willis L.....ALR

CEDAR RAPIDS

Foster, Wayne J.....Higley bldg. ALR
von Lackum, John Kenneth...Merchants Nat'l Bank bldg. ALR

CLINTON

Blackmar, Francis B.
Major (M.C.), Schick General Hospital ALR
Foster, Warren H.....217 Fifth av. S. ALR
Weih, E. P.....217 Fifth av. S. OALR

COUNCIL BLUFFS

Dean, F. W.....22 S. Sixth OALR
Howard, Lloyd G.....Park bldg. OALR
Maiden, Sydner D.....Bennett bldg. OALR

CRESTON

Childs, H. A.....1016 W. Adams OALR

DAVENPORT

Hands, Sidney G.....201 W. 2nd ALR
Harkness, Gordon F.....Putnam bldg. OALR
Hofmann, W. P.....Kahl bldg. OALR
Lamb, Harry Howe.....220 Main ALR
Rock, John Emmett.....Putnam bldg. ALR

DES MOINES

Downing, James A.....Bankers Trust bldg., Z9 ALR
Jones, Cecil C.....Equitable bldg., Z9 OALR
Kirch, Walter.....417 Bankers Trust bldg., Z9 ALR
Lynch, Robert J.....Equitable bldg., Z9 ALR
Matheson, John H.....Equitable bldg., Z9 OP
Merkel, Byron M..Major (M.C.), 802 Equitable bldg., Z9 ALR

DUBUQUE

Connelly, Edgar.....7 Roshek bldg. ALR
Quinn, Francis P.....887 Mt. Carmel road ALR
Thompson, Howard E.....1110 Main OALR
Thorson, John A.....Roshek bldg. OP

FORT DODGE

Chase, S. B.....Carver bldg. OALR
Kluever, Herman C.....Lt. Comdr. (M.C.), Carver bldg. OP

FORT MADISON

Reimers, R. S.....613 Eighth ALR

IOWA CITY

Albright, George C.....104 S. Clinton ALR
Evers, Lorance Bixler.....University Hospital ALR
Leinfelder, Placidus J.....University Hospital OP
Lierle, Dean M.....University Hospital ALR-PI S
O'Brien, C. S.....University Hospital OP
Shipley, J. L.....Capt. (M.C.), Pre-Flight School OALR
Swan, Kenneth C.....University Hospital OP

KNOXVILLE

Boden, Worthey C.....Capt. (M.C.) ALR

LE MARS

Joynt, M. J.....Royal bldg. ALR

IOWA — Continued

MARSHALLTOWN
French, R. F. ..... Masonic Temple **OALR**
Wolfe, Otis R. ..... Masonic Temple **OP**

MASON CITY
Chenoweth, Charlie Everette ..... Forester's bldg. **ALR**
Fallows, Howard D. ..... Forester's bldg. **OALR**
O'Brien, Steve A. ..... First Nat'l Bank bldg. **OALR**

OTTUMWA
Prewitt, Leland Howard ..... Box 452 **ALR**

SHENANDOAH
Maloy, Wayland H. ..... 608½ W. Sheridan av. **OP**

SIOUX CITY
Decker, J. C. ..... Frances bldg., Z8 **OALR**
Dvorak, Joseph E. ..... Davidson bldg., Z13 **OP**
Gittins, T. R. ..... Davidson bldg., Z13 **OALR**
Reeder, James E., Jr.
Capt. (M.C.), 523 Davidson bldg., Z13 **OP**
Reeder, J. E. ..... Davidson bldg., Z13 **OALR**
Tripp, Leroy R. ..... Security bldg., Z8 **OALR**

WATERLOO
Bender, H. A. ..... Black bldg. **ALR**
Joynt, A. J. ..... 525 Sycamore **OALR**
Reuling, F. H. ..... Black bldg. **OALR**
Russell, R. E. ..... 606 Commercial **OALR**

KANSAS

ATCHISON
Brlbach, Evgene J. ..... Professional bldg. **OALR**

COFFEYVILLE
Chadwick, Ira B. ..... 717½ Walnut **OALR**

CONCORDIA
Robertson, Edward N. ..... New Caldwell bldg. **OALR**
Simons, Max ..... Capt. (M.C.), P.O.W. Camp **ALR**

EMPORIA
Granger, W. B. ..... Gazette bldg. **OALR**
Trimble, Clarence S. ..... Gazette bldg. **OALR**

FORT LEAVENWORTH
Rosenbaum, Harry D. ..... Maj. (M.C.), 211 Auger av. **OP**

FORT RILEY
Schlanser, A. E. ..... Col. (M.C.), Post Surgeon's Office **OALR**

HUTCHINSON
Greever, Boyd L. ..... 100 N. Main **OALR**
Quiring, Walter O. ..... 100 First West **ALR**

KANSAS CITY
Billingsley, John A. ..... Huron bldg., Z10 **OP**
Spake, LaVerne B. ..... Brotherhood bldg., Z11 **ALR**

LAWRENCE
Powell, Lyle S. ..... Col. (M.C.), 736 Kentucky st. **OALR**

MANHATTAN
Dyer, B. G. ..... 109½ S. 4th **ALR**

NEWTON
Morgan, Harold Eugene ..... 210 S. Pine st. **OP**

PARSONS
Blasdel, Tyler David ..... 110½ S. 18th **ALR**

PITTSBURG
Haas, Louis Richard ..... Smith Clinic **OALR**

PRATT
Pitman, Will D. ..... First Nat'l Bank bldg. **ALR**

TOPEKA
Allen, George V. ..... 835 Kansas av. **OALR**
Anderson, Orrin E.
Capt. (M.C.), Winter General Hospital **ALR**
Boggs, Frank C. ..... Mills bldg. **OP**
Kirkpatrick, Hazen L., Capt. (M.C.),
1320 Pembroke Lane **ALR**
Mosher, Henry A. ..... Capt. (M.C.), Winter Gen. Hosp. **OP**
Reed, William W. ..... Mills bldg. **ALR**

WICHITA
Brownell, Morton E. ..... 106 W. Douglas, Z2 **OP**
Carter, E. D. ..... Central bldg., Z2 **ALR**
Cheney, James W. ..... Schweiter bldg., Z2 **OALR**
Gillett, Wilbur G. ..... 201½ N. Main, Z2 **OP**
Gsell, J. F. ..... Beacon bldg., Z2 **OALR**
Maggard, D. I. ..... Orpheum bldg., Z2 **OALR**
Marshall, Hal E. ..... 105 S. Broadway, Z2 **ALR**
Reifsneider, J. Stanley ..... Beacon bldg., Z2 **ALR**
Seydell, E. M. ..... First Nat'l Bank bldg., Z2 **ALR**
Tippin, Ernest E. ..... First Nat'l Bank bldg., Z2 **ALR**
Weaver, T. W. ..... 201 N. Main, Z2 **OP**

WINFIELD
Ralls, C. T. ..... State Bank bldg. **OALR**

KENTUCKY

CAMP BRECKENRIDGE
Bornstein, Max. . Maj. (M.C.), Post Hospital, 1570 S. U. **ALR**

CAMP CAMPBELL
Cleary, James A. ..... Major (M.C.), 1580th Service Unit **ALR**

COVINGTON
Bledsoe, R. W. ..... First Nat'l Bank bldg. **OALR**

FORT KNOX
Gans, Jerome A. ..... Lt. (M.C.), 0-535443, Station Hosp. **OP**
Thompson, William R., Lt. Col. (M.C.),
1550 Service Unit, Station Hospital **ALR**

FRANKFORT
Snyder, William S., Jr. ..... 324 W. Main **ALR**

FRANKLIN
Armstrong, R. M. ..... **OALR**

GLASGOW
Weldon, William A. ..... **OP**

HARLAN
Gunn, Milus L. ..... 107 E. Mound st. **ALR**

LEXINGTON
DeWeese, Clarence ..... 201 Second st., Z15 **OALR**
Frey, Walter D. ..... 200 N. Upper st., Z15 **OP**
Marks, Samuel Blackburn ..... 200 W. Second, Z15 **ALR**
Stern, Milton J. ..... First National Bank bldg., Z3 **OP**

KENTUCKY — Continued

LOUISVILLE

Baker, Melvin C.....Heyburn bldg., Z2 OP
Bass, A. L.....Heyburn bldg., Z2 OALR
Dean, Walter.....300 Francis bldg., Z2 OALR
Dulaney, Octavius.....Brown bldg., Z2 ALR
Hall, G. C.....Brown bldg., Z2 OALR
Heitger, J. D.....Heyburn bldg., Z2 OALR
Hutcherson, John Kenneth.....Francis bldg., Z2 OP
Juers, Arthur L.....Heyburn bldg., Z2 ALR
Leggett, Albert E.....614 Fincastle bldg., Z2 OP
Rubright, LeRoy W.
Capt. (M.C.), Nichols General Hospital, Z2 ALR
Victor, Karl N.
1010 Brown bldg., 321 West Broadway, Z2 ALR

LYNCH

Shurtleff, Raymond S.....Box 757 ALR

MADISONVILLE

Garnier, William Hampton.....Doctors bldg. ALR

OWENSBORO

Woolfolk, William L.....115 E. 4th st. ALR

PADUCAH

Abell, Harry Davis.....333 Broadway OP
Pace, Errett.....City Savings Bank bldg. ALR
Reynolds, H. G.....717 Citizens Savings Bank bldg. OALR

LOUISIANA

ALEXANDRIA

Simmonds, Noel T.....1404 Murray OP

BATON ROUGE

Jackson, Rufus.....Louisiana Nat'l Bank bldg., Z6 ALR
McLaurin, James Walter.....251 3rd, Z6 ALR

BARKSDALE FIELD

Henner, Robert.....Capt. (M.C.) ALR
Ridall, Earle G.....Major (M.C.), Station Hospital ALR

CAMP CLAIBORNE

Fishof, Frank E.....Capt. (M.C.), Station Hospital ALR

CAMP LIVINGSTON

Phillips, James R.....Major (M.C.), Station Hospital OP

LAKE CHARLES

Moss, Olin W.....Weber bldg. ALR

NEW ORLEANS

Alexander, Lucian W.....Maison Blanche bldg., Z16 ALR
Bahn, Charles A.....Pere Marquette bldg., Z12 OP
Beil, Wallace C.....812 Maison Blanche bldg., Z16 OP
Benz, Alvin H., Capt. (M.C.),
Station Hospital, N.O.P.E., Jefferson Branch, Z12 OP
Blum, Henry N.....American Bank bldg., Z12 OP
Bradish, Robert F., Col. (M.C.),
Port of Embarkation, Poland and Dauphin sts., Z12 OP
Brewster, H. F.....913 Richards bldg. ALR
Brown, Charles Lafayette.....713-15 Richards bldg. ALR
Buffington, Wiley R.....Hibernia Bank bldg., Z12 OP
Clark, William B.....American Bank bldg., Z12 OP
Cox, Charles L.....Pere Marquette bldg., Z12 ALR
Crebbin, Alexander R.....Maison Blanche bldg., Z16 OP
Deitchman, Louis, Major (M.C.),
182 General Hospital, LaGarde General Hospital, Z12 ALR
Fuchs, Valentine Henry.....American Bank bldg., Z12 ALR
Goldberg, Sol....Major (M.C.), La Garde Hospital, Z12 OP
Gooch, John B.....Canal Bank bldg., Z12 ALR
Granberry, Carl.....Maison Blanche bldg., Z16 ALR
Hanley, Jeanne C. Roeling....Maison Blanche bldg., Z16 ALR
Irwin, John Joseph.....Baronne bldg., Z12 ALR
Kearney, Harold Leslie.....1403 Delachaise, Z15 ALR
LeJeune, F. E.....Ochsner Clinic, Z15 ALR
McComiskey, Arthur James.....3420 Prytania, Z15 ALR
McGrath, Philip R., Lt. Comdr. (M.C.),
U. S. Naval Hospital, Z12 ALR
Mueller, Lawrence Wm.
Maj. (M.C.), LaGarde General Hospital, Z12 ALR
Smith, Victor C.....American Bank bldg., Z12 OP
Taquino, George J.....Canal bldg., Z12 ALR
Wagner, W. A.....200 Carondelet, Z12 ALR
Weil, A. I.....3439 Prytania, Z15 ALR
Wood, Spencer.....500 Verret, Z15 ALR

PINEVILLE

Ewing, Fayette C.....(Retired), "Nosegay."

SHREVEPORT

LaRue, C. L.....Highland Clinic, Z50 OALR
Scales, J. L.....Medical Arts bldg., Z45 OALR
Wilkinson, Joel A.....1130 Louisiana av., Z7 OALR

MAINE

AUGUSTA

Turner, Oliver W.....37 Stone OALR

BANGOR

McQuoid, Robert Moffat.....39 Columbia ALR
Whitworth, John Ewing.....49 Hammond ALR

BATH

Kershner, W. E.....117 Front OALR

FORT WILLIAMS

Irgens, Edwin R.....Maj. (M.C.), Station Hospital ALR

LEWISTON

Chaffers, William H.....190 Bates ALR

PORTLAND

Beach, S. Judd.....704 Congress, Z4 OP
Cummings, George O.....47 Deering, Z4 ALR
Gordon, Charles H.....46 Deering, Z4 ALR
Haskell, Alfred W.....142 High, Z3 OP
Holt, E. E., Jr.....723 Congress, Z4 OP
Johnson, Henry P.....32 Deering, Z3 ALR
McAdams, R.....704 Congress, Z4 OP

WATERVILLE

Hill, Frederick T.....Professional bldg. ALR
Hill, Howard F.....177 Main OP

MARYLAND

ABERDEEN PROVING GROUND

Gouterman, Joseph I.....Maj. (M.C.), Station Hospital OP

ANNAPOLIS

Macnie, John P.
Lt. Comdr. (M.C.), U. S. Naval Hospital OP
Platt, Edward S.
Lt. Comdr. (M.C.), Sick Qtrs., U. S. N. Academy OP
Tiedke, G. E., Lt. Comdr. (M.C.), 602 Dreams Landing OP
Welch, Robert S. G.....86 State Circle OALR

MARYLAND — Continued

BAINBRIDGE

Stewart, James K., Comdr. (M.C.), U. S. Naval Hosp. ALR
Wible, LeRoy E.....Lt. Comdr. (M.C.), Port Deposit ALR
Young, Charles A.....Comdr. (M.C.), U. S. Naval Hosp. OP

BALTIMORE

Bagley, Cecil Hopkins.....2 East Read, Z2 OP
Bodenheimer, Ernst.....1212 Eutaw Place, Z17 OP
Bordley, James Jr.....4 Charlcote Place, Z18 OALR
Breitstein, M. Lewis.....1213 Eutaw Place, Z17 ALR
Clapp, C. A.....513 N. Charles st., Z1 OP
Dix, Harold C.....405 N. Charles st., Z1 OP
Fox, Samuel L.....1820 Eutaw pl., Z17 ALR
Friedenwald, Harry.....1212 Eutaw pl., Z17 OP A
Friedenwald, J. S.....1212 Eutaw pl., Z17 OP
Goldback, L. J.....6 East Eager, Z2 OP
Goldberg, Herman Krieger.....8 E. Eager st., Z2 OP
Guyton, Jack Smallwood.....John Hopkins Hospital, Z5 OP
Haines, Henry L.....Johns Hopkins Hospital, Z5 ALR
Hughes, William F. Jr.....61 9 N. Washington st., Z5 OP
Kemler, Joseph I.....1908 Eutaw pl., Z17 OALR
Looper, Edward Anderson.....104 W. Madison, Z1 OALR
McKenzie, William Raymond.....101 W. Read, Z1 ALR
MacLean, Angus L.....1201 N. Calvert, Z2 OP
Maumenee, Alfred E.....Johns Hopkins Hospital, Z5 OP
Randolph, M. Elliot..Maj. (M.C.), 4302 Rugby rd., Z10 OP
Raskin, Moses.....2038 Eutaw pl., Z17 OALR
Scholz, Roy O.....Johns Hopkins Hospital, Z5 OP
Small, Mary L.....18 W. Read, Z1 OP
Smith, Leslie M.
Lt. Comdr. (M.C.), U. S. Marine Hospital, Z11 OP..
Tarun, William.....104 W. Madison, Z1 OP
Woods, Alan C.....Johns Hopkins Hospital, Z5 OP
Zinn, Waitman Farnsworth.....Medical Arts bldg., Z1 ALR

BETHESDA

Baker, Daniel Clifton, Jr., Lt. (M.C.),
U. S. Naval Hopital, Z14 ALR
Town, A. E., Lt. Comdr. (M.C.),
U. S. Naval Hospital, Z14 OP
Troup, Wilson J., Lt. Comdr. (M.C.),
U. S. Naval Hospital, Z14 ALR

CHEVY CHASE

Rice, Carl E.....6500 Maple av., Z15 OP

FOREST GLEN

Ridout, Geo. B.....Lt. Comdr. (M.C.), U.S.N. ALR

FORT GEORGE MEADE

Philpott, I. W...Maj. (M.C.), General Hospital No. 29 ALR

FRUITLAND

Daisey, Willard Orville..... ALR

GREENBELT

Edmunds, Stanley L.
Lt. (M.C.), U.S.N.R., 13-D Hillside bldg. ALR

HAGERSTOWN

Smith, W. Hamilton.....314 Wareham bldg. ALR

HYATTSVILLE

Goad, R. R.....Lt. Comdr. (M.C.), 4211 Farragut av. OP

SALISBURY

Gillis, Marion Howard, Jr., Maj. (M.C.),..405 Bush st. OP

MASSACHUSETTS

ARLINGTON

Carroll, Walter J. E.....5 Chestnut, Z74 ALR

BELMONT

Cogan, David G.....30 Clark st. OP

BOSTON

Allman, C. H., Comdr. (M.C.), U.S.N. Receiving Station ALR
Carter, Albert A.....419 Boylston, Z16 OP
Casten, Virgil G.
Lt. Comdr. (M.C.), 520 Commonwealth av., Z15 OP
Chandler, Paul A.....5 Bay State rd., Z15 OP
Chenery, W. E.....377 Commonwealth av., Z15 ALR
Chisholm, Julian F., Jr.....204 Devonshire st., Z10 OP
Cline, Samuel.....416 Marlboro, Z15 ALR
Cowan, Clyde R.....69 Bay State rd., Back Bay, Z15 ALR
DeBlois, Elizabeth.....395 Commonwealth av., Z15 ALR
Donahue, Hugh C.....520 Commonwealth av., Z15 OP
Dunphy, Edwin B.
Lt. Comdr.(M.C.), 243 Charles st., Z14 OP
Easton, Mahlon T.....264 Beacon st., Z16 OP
Evans, Maurice George.....416 Marlboro, Z15 ALR
Fein, Harry.....483 Beacon st., Z15 ALR
Fineberg, Nathan L.....475 Commonwealth av., Z15 ALR
Fred, Gustave B.....520 Beacon, Z15 ALR
Freedman, L. M.....475 Commonwealth av., Z15 ALR
Garland, Frederick E.....483 Beacon, Z15 LR
Goodale, Robert L.....330 Dartmouth, Z16 ALR
Goodall, Edwin Baker.....71 Bay State rd., Z15 OP
Gorney, Arthur J., Capt. (M.C.).....230 Boylston st. ALR
Haley, William Thomas.....531 Beacon, Z15 ALR
Happ, L. C.....280 Beacon st., Z16 ALR
Holmes, Edgar M.....330 Dartmouth, Z16 ALR
Hoover, Walter Boyd.....605 Commonwealth av., Z15 ALR
Inglis, Harry J.....43 Bay State rd., Z15 ALR
Johns, Juanita Pearl.....313 Commonwealth av., Z15 OP
Johnson, Leighton F.....29 Bay State rd., Z15 ALR
Kazanjian, V. H.....475 Commonwealth av., Z15 LR-PI S
Kelley, Vincent J.....520 Commonwealth av., Z15 ALR
Kleinert, Margaret N.....1101 Beacon st. ALR
Lancaster, W. B.....520 Commonwealth av., Z15 OP
Lovesey, Burton E.....76 Bay State rd., Z15 ALR
Lurie, Moses H.....483 Beacon, Z15 ALR
Meltzer, Philip E.....20 Charlesgate West, Z15 ALR
Messenger, Harry K.....313 Commonwealth av., Z15 OP
Mills, Chester R.....520 Commonwealth av., Z15 ALR
Mueller, Werner.....412 Beacon, Z15 ALR
Phillips, James Winston
Lt. (M.C.), Camp Miles Standish, Port of Embarkment ALR
Poirier, George H.....60 Bay State rd., Z15 ALR
Porter, Charles T.....520 Commonwealth av., Z15 ALR
Quincy, J. E.....270 Commonwealth av., Z16 ALR
Regan, James J.....520 Commonwealth av., Z15 OP
Regan, William F.....19 Bay State rd., Z15 ALR
Richardson, John R.....395 Commonwealth av., Z15 ALR
Rowland, W. D.....84 Commonwealth av., Z16 OP
Sachs, Benjamin.....520 Beacon, Z15 OP
Schall, LeRoy A.....243 Charles st., Z14 ALR
Skirball, J. J.....353 Commonwealth av., Z15 OP
Sloane, Albert E.....416 Marlborough, Z15 OP
Terry, Theodore L.....140 Marlborough, Z16 OP
Thompson, Peter H.....308 Commonwealth av., Z15 OP
Thorne, Fred S.....270 Commonwealth av., Z16 OP
Tingley, Louisa Paine.....416 Marlborough, Z15 OP
Tobey, George L., Jr.....403 Commonwealth av., Z15 ALR
Verhoeff, Frederick.....395 Commonwealth av., Z15 OP

MASSACHUSETTS — Continued

Wells, David W.....31 Bay State rd., Z15 OP
Wilkins, Samuel Henry....270 Commonwealth av., Z16 OP

BROCKTON
Jones, Edward Earl.....141 West Elm, Z8 ALR
Nerbonne, Joseph J.....231 Main, Z1 OP

BROOKLINE
Gillespie, Elmer H.....1101 Beacon, Z46 ALR

CAMBRIDGE
Butler, Edmund John.....25 Garden st., Z38 ALR

CHELSEA
Lane, Franklin F.
Capt. (M.C.), U. S. Naval Qts. "H", Z50 OALR

CHICOPEE FALLS
Dearmin, R. M.
Major (M.C.), Station Hospital, Westover Field ALR

CLINTON
Monahan, J. A.....181 Chestnut OALR
O'Toole, Francis Austin.....181 Chestnut ALR

FALL RIVER
Ryder, D. R.....151 Rock ALR

FITCHBURG
Quinlan, Joseph D.....44 Prichard st. ALR

FORT DEVANS
Riseman, Benjamin....Capt. (M.C.), Lovell Gen. Hosp. ALR
Schultz, Abraham....Capt. (M.C.), Lovell Gen. Hospital OP

FRAMINGHAM
Berger, Murray....Capt. (M.C.), Cushing Gen. Hospital ALR
Page, Robert O., Major (M.C.),
Cushingham General Hospital ALR
Salis, Frederick H.....132 Union av. OP
Tomb, Everett H.....Maj. (M.C.), 46 Lexington ALR

GARDNER
Bone, Herman D.....19 Pleasant OALR

GLOUCESTER
Odeneal, Thomas H.....No. 9 Middle OALR

GREENFIELD
Millett, Frank A.....40 High OALR
Powers, John T. H.....57 Federal st. ALR

HAVERHILL
Birdsall, Clarence H.....30 Summer ALR
Capeles, T. F.....191 Merrimack ALR
Kapp, Henry.....50 Merrimack ALR
Shlossberg, Frank Ronald.....116 Emerson ALR
Trull, Alfred C.....3 Washington square OP

HOLYOKE
Burke, John R.....207 Elm OALR
Halton, Edward P.....1456 Northampton st. ALR

LAWRENCE
Byrne, Harry V.....301 Essex OP

LOWELL
Berman, Philip G.....174 Central OP
Leahey, Brendan D.....9 Central OP
Leahey, George A.....45 Merrimack OP
Wiggin, William Irving.....202 Central ALR

LYNN
Jones, E. W.....44 Atlantic OP A
McIntire, Frederic J.....63 N. Common ALR
O'Reilly, W. F.....44 S. Common OALR
Rafferty, George E.....173 N. Common ALR

MALDEN
French, Robert Weller.....181 Pleasant OP
Haire, Paul G.....6 Pleasant, Z48 OP

MARBLEHEAD
Mosher, Harris P.....127 Front ALR

MEDFORD
Haley, Paul J. D.....338 Winthrop ALR

MELROSE
DeWolf, Charles W.....8 Porter ALR

NORTHAMPTON
Collins, J. D.....187 Main ALR
Dow, Frank E.....4 West OP
Fackler, C. L., Lt. Comdr. (M.C.), USNR
U. S. Naval Reserve School OALR
Seelig, Charles A.
Lt. (M.C.),, U.S.N.R., 10 Dryads Green ALR

PALMER
Moore, G. A.....Box 471 OALR

PITTSFIELD
Porter, Byron H.....18 Bank Row ALR
Thompson, G. H.....74 North OALR

QUINCY
Donovan, Richard Joseph.....1400 Hancock st., Z69 OALR
Gilmore, Robert O.....1159 Hancock ALR

SALEM
Adams, John G.....24 Winter ALR
Hennessey, W. W.....333 Essex OALR
Tolman, Henry.....136 Federal ALR

SOMERVILLE
Solo, Adrian.....174 Highland av. ALR

SPRINGFIELD
Boss, Eugene George.....1537 Main, Z3 OALR
Byrnes, Harry F.....67 Chestnut, Z5 OALR
Gaudreau, Honore E.....293 Bridge, Z3 OP
Goodell, William.....121 Chestnut, Z3 ALR
Irwin, Vincent J., Jr.....1634 Main, Z3 OALR

TAUNTON
Cowan, Thomas H.
Capt. (M.C.), Station Hosp., Camp Miles Standish OP

WALTHAM
Mackenzie, Roland C.....30 Grant, Z54 OP

WELLESLEY HILLS
Richards, Lyman G., Lt. Comdr. (M.C.), USNR
15 Whiting Road ALR

MASSACHUSETTS — Continued

WILLIAMSTOWN

Cetner, John A., Lt. (M.C.), U. S. Navy Flight Preparatory School OP

WORCESTER

Berry, Gordon....36 Pleasant, Z8 ALR
Caron, Armand L....36 Pleasant, Z8 ALR
Croissant, Charles A....390 Main, Z8 ALR
Cross, A. E....390 Main, Z8 OP
Gibby, Harold James....27 Elm, Z8 ALR
Goodspeed, Harry B....36 Pleasant, Z8 ALR
Holzer, William F....36 Pleasant, Z8 OP
Lewis, James P., Comdr. (M.C.), Worcester Polytechnic Institute ALR
Messier, A. Eugene....390 Main, Z8 OALR
Murphy, William E....390 Main, Z8 ALR
Rice, John E....390 Main, Z8 OALR
Tegelberg, Julius J. Lt. Comdr. (M.C.), 12 Monterey rd., Z6 ALR
Whitney, Percy T....390 Main, Z8 OP

MICHIGAN

ANN ARBOR

Barr, Albert S....First Nat'l Bank bldg. OP
Falls, Harold Francis....University Hospital OP
Fralick, F. Bruce....First Nat'l Bank bldg. OP
Furstenberg, A. C....First Nat'l Bank bldg. ALR
Maxwell, James H....First Nat'l Bank bldg. ALR
Teed, R. Wallace, Lt. Comdr. (M.C.), 410 Highland rd. ALR

BATTLE CREEK

Barkhorn, Charles W., Lt. Col. (M.C.), Percy Jones General Hospital ALR
Cusick, Paul L., Maj. (M.C.), Percy Jones General Hosp. OP
Fraser, R. H....25 W. Michigan av. ALR
Haughey, Wilfrid H....Post bldg. OALR
Krimsky, Emanuel....Maj. (M.C.), Percy Jones Hospital OP
Stewart, Charles E., Capt. (M.C.) Percy Jones Gen. Hosp. ALR
Wencke, Carl G....1015 Security Tower ALR

BAY CITY

Baker, Charles H....Bay City Bank bldg. OALR
Criswell, Robert Harold....Phoenix bldg. OP
Kessler, Mana....311 Center av. ALR
Sherman, R. Newton....Phoenix bldg. OALR

BENTON HARBOR

Cawthorne, Harold Jesse....239 Pipestone ALR
Westervelt, H. O....239 Pipestone ALR

BIRMINGHAM

Ferris, Ralph G....55 W. Maple av. ALR

COLDWATER

Thomas, James Alanson, Southern Michigan Bank bldg. ALR

DETROIT

Amberg, Emil....1244 W. Boston blvd., Z2 ALR
Barnett, Saul E....25 Parsons, Z1 ALR
Beattie, Robert....David Whitney bldg., Z26 OALR
Begle, Howell L....2730 E. Jefferson av., Z7 OP
Bentley, Neil I....David Whitney bldg., Z26 OALR
Brown, Audrey O....5057 Woodward av., Z2 OALR
Brunk, Clifford F....7815 E. Jefferson av., Z14 ALR
Campbell, Duncan A....David Whitney bldg., Z26 OALR
Carter, John M....David Whitney bldg., Z26 ALR
Carter, Leland F....David Whitney bldg., Z26 OP
Clarke, George L....910 Chalmers, Z15 ALR
Clifford, T. P....David Whitney bldg., Z26 OALR
Cohoe, Don A....13535 Woodward av., Z3 ALR
Cooper, Edmond L....414 David Whitney bldg., Z26 OP
Crossen, Henry F....David Whitney bldg., Z26 ALR
Croushore, James E....Maj. (M.C.), 2045 Edison, Z6 ALR
Defnet, William A....28 Adams av., West, Z26 ALR
Dill, J. Lewis....Henry Ford Hospital, Z2 ALR
Dunlap, Henry Arthur....7815 E. Jefferson av., Z14 OP
Gemeroy, Joseph C....Henry Ford Hospital, Z2 OALR
Gleason, John E....David Whitney bldg., Z26 ALR
Glowacki, Ben Francis....Maccabee bldg., Z2 OALR
Gonne, William S....David Whitney bldg., Z26 ALR
Goux, Raymond Salot....David Whitney bldg., Z26 OALR
Hale, Arthur Stanley....Eaton Tower, Z26 OALR
Hammond, Arthur....David Whitney bldg., Z26 ALR
Harrell, Voss....Hofman bldg, Z1 ALR
Hartgraves, Hallie....28 West Adams av., Z26 OP
Heath, Parker....David Whitney bldg., Z26 OP
Hughes, Ray W....David Whitney bldg., Z26 OALR
Joinville, Euclide V....Stroh bldg., Z26 ALR
Keating, T. F....Maccabee bldg., Z2 OALR
Lamberson, Frank Abrom....19600 Grand River, Z23 ALR
Lathrop, Frank D....Maj. (M.C.), 103 Moran rd., Z30 ALR
Lepard, Cecil W....David Whitney bldg., Z26 OP
McCullough, Lester E....1553 Woodward av., Z26 OP
MacFarlane, Howard W....David Whitney bldg., Z26 ALR
Mercer, R. E....126 Monterey, Z3 LR
Mond, Edward....Eaton Tower, Z26 ALR
Morton, J. B....90 E. Warren av., Z1 OALR
Munson, F. T....2201 E. Jefferson av., Z7 ALR
O'Hora, James Timothy....General Motors bldg., Z2 ALR
Parker, Walter R....David Whitney bldg., Z26 OP
Peirce, Howard W....5181 Van Dyke av., Z13 ALR
Pino, Ralph H....David Whitney bldg., Z26 OP
Pittman, John E....David Whitney bldg., Z26 OP
Poos, E. E....Fisher bldg., Z2 OALR
Potter, Willis A....7450 Grand River av., Z4 ALR
Proctor, Bruce....Eaton Tower, Z26 ALR
Robb, James Milton....David Whitney bldg., Z26 ALR
Ryerson, F. L....Fine Arts bldg., Z26 OALR
Shurly, Burt R....62 Adams av. West, Z26 ALR
Simpson, H. Lee....David Whitney bldg., Z26 ALR
Summers, William S....David Whitney bldg., Z26 OALR
Sutherland, Jacob Marion....Fisher bldg., Z2 ALR
Vossler, Albert E....David Whitney bldg., Z26 OALR
Weaver, Delmar Franklin....Henry Ford Hospital, Z2 ALR
Wendel, J. S....David Whitney bldg., Z26 ALR
Whitney, E. L....Henry Ford Hospital, Z2 OP
Wilkinson, A. P....3001 W. Grand blvd., Z2 ALR
Woodry, Norman Lee....10 Witherell, Z26 ALR
Woodworth, William P....2501 W. Grand blvd., Z8 OALR

FLINT

Hubbard, Wm. B....302 Paterson bldg., Z3 OALR
Smith, Eugene C....Union Industrial bldg., Z3 OALR
Stevenson, William W....Kresge bldg., Z3 OALR
White, Herbert T....National bldg., Z3 OP

FORT BRADY

Good, Richard P....Maj. (M.C.), Station Hospital ALR

FORT CUSTER

Hauser, Maurice J....Maj. (M.C.), Station Hosp. OP
Von Zelinski, W. F., Col. (M.C.)....Headquarters OP

MICHIGAN — Continued

GRAND RAPIDS
Dean, Alfred .....Medical Arts bldg., Z2 OP
Dewar, Murray M.....Medical Arts bldg., Z2 OP
Doran, Frank.....Metz bldg., Z2 ALR
Gilbert, Ralph H.....Metz bldg., Z2 OP
Heetderks, D. R.....Medical Arts bldg., Z2 ALR
Laird, Robert G.....114 E. Fulton st., Z2 OP
McRae, J. H.....Medical Arts bldg., Z2 OP
Moll, Arthur M.....Powers Theatre bldg., Z2 ALR
Phillips, James William.....Medical Arts bldg., Z2 OP
Smith, Ferris.....Blodgett Medical bldg., Z6 ALR-PI S.
Snapp, Carl F.....Medical Arts bldg., Z2 ALR

GROSSE POINT
Merkel, Charles C.....85 Kercheval av., Z30 ALR

HOLLAND
DeVries, Harold G.....36 East 8th ALR

IONIA
Sedgwich, O. W..... OALR

IRON MOUNTAIN
Boyce, G. H.....First Nat'l Bank bldg. OALR
Menzies, Clifford G.....733 Woodward av. ALR

JACKSON
Hardie, George C.....290 W. Michigan av. OALR
McGarvey, William Edward....Jackson City Bank bldg. OALR
Newton, Ray E.....1010 Reynolds bldg. OP
Winter, George E.....117 W. Michigan av. OALR

KALAMAZOO
Fast, Ralph B.....136 E. Michigan av., Z4 ALR
Fulkerson, C. B.....107 W. Michigan av., Z9 OALR
Grant, Fred E.....Kalamazoo Nat'l Bank bldg., Z9 OALR
Wilbur, Edward P.....American Nat'l Bank bldg., Z4 OALR

LANSING
Behen, William C.....535 S. Capitol av., Z15 ALR
Burdick, Austin F.....P. O. Drawer 1378, Z4 OALR
Ellis, C. Ward.....215 N. Walnut, Z15 ALR
Wetzel, John O.....Olds Tower bldg., Z8 OP

MENOMINEE
Jones, William S.....521 Sheridan road OALR

MIDLAND
Pike, Melvin H.....Reinhart bldg. OALR

MT. CLEMENS
Croman, Joseph M., Jr.....115 Cass av. OALR

MUSKEGON
Loder, L. L...506 Hackley Union Nat'l Bank bldg., Z11 OP
Mandeville, Clifford Bagley.....Hackley Union Bank ALR

NILES
Moore, Thomas Scott.....107 N. 2nd OP

OWOSSO
Pochert, Rolland C.....Matthews bldg. OALR

PONTIAC
Larson, Bertil T.....Riker bldg., Z15 ALR
Riker, Aaron D.....Riker bldg., Z15 OP

PORT HURON
Fraser, Robert.....940 Military ALR

ST. JOSEPH
McDermott, John J.....505 Pleasant st. OALR

SAGINAW
Cady, F. J.....402 S. Jefferson av. OALR
Cortopassi, Andre J.....324 S. Washington av. ALR
Gomen, Louis D.....Eddy bldg. OP
Howell, Donald M.....Wiechmann bldg. ALR
McKinney, A. R.....330 S. Washington av. OP
Richter, Harry J., Lt. (M.C.).....2305 Adams blvd. ALR

SELFRIDGE FIELD
Calvert, Raymond R.....Maj. (M.C.), Station Hosp. OP
Sims, J. Lawrence, Capt. (M.C.).....Station Hospital ALR

MINNESOTA

ALBERT LEA
Folken, Frank Garrett.....201 S. Broadway OALR

BEMIDJI
Garlock, Arthur Varney.....217½ 3rd OP

CROOKSTON
Morely, George A.....P. O. Box 452 OP
Oppegaard, C. L.....220 S. Broadway ALR
Parsons, J. G.....Crookston Clinic OALR

DULUTH
Armstrong, E. Leslie.....Medical Arts bldg., Z2 ALR
Athens, Alvin G.....Medical Arts bldg., Z2 OP
Hilding, Anderson C.....Medical Arts bldg., Z2 OALR
Knapp, F. N.....Medical Arts bldg., Z2 OALR
Olson, Archie.....Medical Arts bldg., Z2 OP
Shastid, T. H.....202 W. Superior., Z2 OP
Tilderquist, D. L.....Medical Arts bldg., Z2 OALR
Winter, John A.....205 W. 2nd, Z2 OALR

FERGUS FALLS
Combacker, Leon C.....101½ Lincoln av. W. OP

FORT SNELLING
Downing, Arthur H...Capt. (M.C.), Med. Ind. Sta., Z11 OP

HIBBING
Morsman, L. W.....Morsman Clinic OP
Sinamark, Andrew.....Power bldg. OALR

MINNEAPOLIS
Boies, Lawrence R.....Medical Arts bldg., Z2 ALR
Borgeson, E. J.....Medical Arts bldg., Z2 OP
Cabot, C. M.....Medical Arts bldg., Z2 ALR
Camp, Walter E.....Medical Arts bldg., Z2 OALR
Clark, H. S.....2615 Park av., Z7 OALR
Curtin, John F.....Medical Arts bldg., Z2 ALR
Dahl, Elmer O.....Medical Arts bldg., Z2 ALR
Dorge, Richard Irving.....1111 Nicollet av., Z2 OALR
Fink, Leo W.....Medical Arts bldg., Z2 ALR
Fink, Walter H.....Medical Arts bldg., Z2 OP
Garten, Joseph Lambert.....Medical Arts bldg., Z2 ALR
Hansen, Erling W.....Medical Arts bldg., Z2 OP
Hoffman, Walter Lees.....Medical Arts bldg., Z2 OP
Howard, William H.
Capt. (M.C.), 3949 Portland av., Z7 OALR

MINNESOTA — Continued

Hymes, Charles.....Medical Arts bldg., Z2 **OP**
Koller, Hermann M.....Medical Arts bldg., Z2 **OP**
Koller, Louis R.....Medical Arts bldg., Z2 **ALR**
Lindberg, Vernon Leslie
Capt. (M.C.), Physicians and Surgeons bldg. **OP**
Litchfield, J. T.....Medical Arts bldg., Z2 **OALR**
Loomis, E. A.....Medical Arts bldg., Z2 **OALR**
McGeary, George E.....Medical Arts bldg., Z2 **ALR**
Macnie, J. S.....Medical Arts bldg., Z2 **OALR**
Matthews, Justus.....Medical Arts bldg., Z2 **LR**
Newhart, Horace.....Medical Arts bldg., Z2 **ALR**
Oberg, C. M.....Syndicate bldg., Z2 **OALR**
Patterson, W. E.....Medical Arts bldg., Z2 **OALR**
Pfunder, Malcom C.....Medical Arts bldg., Z2 **ALR**
Phelps, Kenneth A.....Medical Arts bldg., Z2 **OALR**
Pratt, Fred J.....Physicians and Surgeons bldg., Z2 **OALR**
Ricketts, Floyd B.....Veterans Hospital, Z6 **ALR**
Sandt, Karl E...Capt. (M.C.), 3328 Humboldt av. S., Z8 **OP**
Schwartz, Virgil Joseph.....Medical Arts bldg., Z2 **ALR**
Smith, Arthur E.....Medical Arts bldg., Z2 **OALR**
Stanford, Charles E.....Medical Arts bldg., Z2 **OP**
Strout, G. Elmer.....1009 Nicollet av., Z2 **OALR**
Tangen, George M.....University of Minnesota, Z14 **ALR**
White, C. H..Lt. Comdr. (M.C.), Naval Aviation Base, Z6 **ALR**

NEW ULM

Fritsche, Theodore R.....Fritsche bldg. **OALR**
Reineke, George F.....Fritsche bldg. **OALR**

RED WING

Brusegard, James F.....414 Main **OP**

ROCHESTER

Bair, Hugo L.....Mayo Clinic **OP**
Benedict, William L.....Mayo Clinic **OP**
Brown, Henry A.....937 Sixth av. S. E. **ALR**
Figi, Frederick A.....Mayo Clinic **LR-PI S**
Hallberg, Olav Erik.....Mayo Clinic **ALR**
Havens, Fred Z.....Mayo Clinic **ALR-PI S**
Hempstead, Bert E.....Mayo Clinic **ALR**
Hogan, Chester F., Lt. Comdr. (M.C.), Mayo Foundation **ALR**
Lillie, Harold I.....Mayo Clinic **ALR**
New, Gordon B.....Mayo Clinic **L-PI S**
Prangen, Avery de H.....Mayo Clinic **OP**
Rucker, C. W.....Mayo Clinic **OP**
Wagener, Henry P.....Mayo Clinic **OP**
Williams, Henry Lane.....Mayo Clinic **ALR**

ST. CLOUD

Gaida, Joseph Benedict.....St. Mary's bldg. **OP**
Wenner, W. T.....St. Mary's bldg. **OP**

ST. PAUL

Binger, H. E.....Lowry Medical bldg., Z2 **OALR**
Boeckmann, Egil.....Lowry Med. bldg., Z2 **OALR**
Brown, John C.....Medical Arts bldg., Z2 **OALR**
Burch, Frank E.....754 Linwood Place, Z5 **OP**
Connor, Charles E.....Lowry Medical bldg., Z2 **ALR**
Edwards, Thomas J.....330 Lowry Medical bldg., Z2 **OP**
Fogarty, Charles W.....372 St. Peter, Z2 **OALR**
Grant, H. W.....Lowry Medical bldg., Z2 **OP**
Hochfilzer, John J.....Lowry Medical bldg., Z2 **ALR**
Holmen, Robert W.....1261 Lowry Med. Arts bldg., Z2 **ALR**
Kennedy, William A.....372 St. Peter st., Z2 **OP**
Larsen, Carl L.....Lowry Medical bldg., Z2 **OALR**
Leavenworth, R. O.....Lowry Medical bldg., Z2 **OP**
Nelson, Louis A.....Lowry Medical bldg., Z2 **OP**
O'Reilley, Bernard E.....Lowry Medical bldg., Z2 **OP**
Rothchild, Harold J.....Lowry Medical bldg., Z2 **OALR**
Tracht, Robert R.....Lowry Medical bldg., Z2 **OP**
Wheeler, Merritt W.....Lowry Medical bldg., Z2 **OALR**
Wold, K. C.....Lowry Medical bldg., Z2 **OALR**

WINONA

Loomis, George L.....310 Choate bldg. **ALR**

WORTHINGTON

Stanley, C. R.....508 Tenth **ALR**

MISSISSIPPI

CAMP McCAIN

Knisely, Alan DeG.....Major (M.C.), Station Hospital **ALR**

CLARKSDALE

Wilkins, Ellis LeRoy.....McWilliams bldg. **ALR**

COLUMBUS

Thompson, James P.
Capt. (M.C.), C.A.A.B., Station Hospital **OP**

CORINTH

Hamrick, DeWitt.....601 Jackson **ALR**

GREENVILLE

Montgomery, D. Cameron.....Weinberg bldg. **ALR**

GREENWOOD

Pegues, James Cary.....301 Howard st. **ALR**

GRENADA

Caruthers, Samuel Brooks.....Grenada Clinic **OALR**

GULFPORT

McWilliams, Charles A.....1300 27th av. **ALR**
Rouse, H. K., Jr.
Major (M.C.), McWilliams Clinic, Box 174 **OP**
Wilson, Alfred.....Major (M.C.), 1518 20th av. **OP**

JACKSON

Adkins, George E.....Jackson Infirmary, Z107 **ALR**
Anderson, Ross Epting.....Standard Life bldg., Z27 **ALR**
Cutler, Norman L.
Lt. Col. (M.C.), Veterans Administration **OP**
Hagaman, Van Dyke.....Lamar Life bldg., Z2 **ALR**
Harris, Robin.....964 N. State, Z6 **ALR**
Hollowell, Frederick D.....Lamar Life bldg. **ALR**
Hughes, William Lauch.....Lamar Life bldg., Z2 **ALR**
Linton, Clair S..Maj. (M.C.), Foster Gen. Hosp., Z115 **ALR**
Weinman, Edward B., Capt. (M.C.),
Foster General Hospital, Z115 **OP**

KEESLER FIELD

McAlpine, P. T.....Capt. (M.C.), Station Hospital **OP**
Vanzant, Thomas J.....Lt. (M.C.), Station Hospital **OP**

McCOMB

Cotten, Willis Frank.....202 State st. **ALR**

NATCHEZ

Gaudet, Lucien S.....405 Franklin **OALR**

OXFORD

Guyton, B. S.....512 Van Buren av. **OALR**

VICKSBURG

Edwards, C. J.....First Nat'l Bank bldg. **OALR**
Jones, Edley H.....1301 Washington **OALR**

MISSOURI

CAMP CROWDER

Greenberg, Maynard M.....Maj. (M.C.), Station Hospital OP
Johnson, Malcolm R.....Lt. Col. (M.C.), Station Hosp. ALR

CAPE GIRARDEAU

Tygett, Glenn J.....215 N. Spring ALR

CARTHAGE

Powers, Everett.....305 S. Main OALR

COLUMBIA

Sneed, Carl M.....Exchange bldg. OALR

FORT LEONARD WOOD

Arbuckle, Millard F.....Col. (M.C.), Station Hospital ALR
Simons, Leander T.....Lt. Col. (M.C.), Station Hospital OP

HANNIBAL

Hornback, George A.....500 Broadway ALR

JEFFERSON BARRACKS

Stuard, Charles G.....Major (M.C.), Station Hospital OP

JEFFERSON CITY

Stauffer, Harry B.....234 Madison ALR

KANSAS CITY

Allen, Charles E.....Waldheim bldg., Z6 OALR
Altringer, A. N.....Professional bldg., Z6 ALR
Beil, J. Wallace.....Bryant bldg., Z6 OALR
Black, W. Byron.....Professional bldg., Z6 ALR
Bower, Richard L.....1103 Grand av., Z6 ALR
Connell, Evan Shelby.....Commerce bldg., Z6 OP
Curdy, R. J.....1102 Grand av., Z6 OP
Curran, Desmond
Lt. (M.C.), Federal Reserve Bank bldg., Z6 OP
Curran, E. J.....Federal Reserve Bank bldg., Z6 OP
Curran, Kevin.....Federal Reserve Bank bldg., Z6 OP
Dixon, O. Jason.....Professional bldg., Z6 ALR
Eubank, Ambrose E.....Bryant bldg., Z6 OALR
Foster, Hal.....(Retired), Hyde Park Hotel, Z2 ALR
Fox, C. H.....Schuyler Hotel, 1017 Locust st. OALR
Gilliland, O. S.....Professional bldg., Z6 ALR
Howard, J. W.....(Retired), Residence 6026 McGee, Z2 OALR
Howard, John Clair
Lt. (j.g.) (M.C.), Professional bldg., Z6 ALR
Keith, W. E.....Professional bldg., Z6 OALR
Lemoine, A. N.....Rialto bldg., Z6 OP
Lux, Paul.....Bryant bldg., Z6 ALR
McAlester, Andrew W., Jr.....Bryant bldg., Z6 OP
McKee, Joseph W.....Professional bldg., Z6 OP
Miller, Hugh.....Argyle bldg., Z6 OALR
Myers, John L.....Shukert bldg., Z6 OALR
Roberts, S. E.....Professional bldg., Z6 ALR
Shoemaker, Ferdinand.....3232 Harrison, Z3 OALR
Simpson, M. B.....Professional bldg., Z6 ALR
Teall, Raymond E.....Professional bldg., Z6 ALR
Thomason, H. E.....Professional bldg., Z6 ALR
Yazel, Herman E.....Rialto bldg., Z6 OALR

KIRKWOOD

McQuiston, Ralph J.....Lt. Col. (M.C.), 203 Barter av. ALR

ROLLA

Boiler, W. F., Major (M.C.),
0-197861, 603 E. 10th st. OALR

ST. JOSEPH

Minton, William H.....Corby bldg., Z9 OALR

ST. LOUIS

Alden, Arthur M.....Frisco bldg., Z1 ALR
Alvis, Bennett Y.....7011 Washington blvd., Z5 OP
Bardenheier, F. G. A.....Frisco bldg., Z1 ALR
Beisbarth, Carl.....3720 Washington blvd., Z8 OP
Birsner, Louis J.....5344 Englewood Pl., Z21 ALR
Bisno, Daniel.....4500 Olive st., Z8 OP
Black, W. D.....Metropolitan bldg., Z3 ALR
Boemer, L. C., Maj.. (M.C.), U.S.A.,
3720 Washington blvd., Z8 ALR
Brown, Clyde O.....3720 Washington blvd., Z8 ALR
Bryan, James H.....508 N. Grand blvd., Z3 OP
Bryan, William T. K.....3720 Washington blvd., Z8 ALR
Burns, S. S.....Missouri Theatre bldg., Z3 ALR
Charles, Joseph W.....Humboldt bldg., Z3 OP
Costen, J. B.....Beaumont Medical bldg., Z8 ALR
Dean, Lee W., Jr.....3720 Washington blvd., Z8 ALR
Dobbs, Otto Ross
Comdr. (M.C.), 9927 Riverview drive, Z15 ALR
Drews, Leslie C.....516 Metropolitan bldg., Z3 OP
Dyer, C. P.....Humboldt bldg., Z3 OP
Eber, Carl Theodore.....Carleton bldg., Z1 OP
Elz, Jule Theodore.....3606 Gravois blvd., Z16 OP
Fineberg, Maxwell.....607 North Grand blvd., Z3 ALR
Gissy, C. J.....717 Metropolitan bldg., Z3 OP
Glick, Harry N.....3115 S. Grand blvd., Z18 ALR
Gorrilla, Laurence Vincent.....3115 S. Grand blvd., Z18 OP
Green, John.....3720 Washington blvd., Z8 OP
Gundelach, C. A.....University Club bldg., Z3 ALR
Hansel, French Keller.....634 N. Grand av., Z3 ALR
Hardesty, J. F.....Missouri Theatre bldg., Z3 OP
Hardy, W. F.....Humboldt bldg., Z3 OP
Hildreth, H. Rommel.....Metropolitan bldg., Z3 OP
Hobart, Carl.....Metropolitan bldg., Z3 OP
Hooss, C. H. A.....3115 S. Grand blvd., Z18 OP
Hourn, George E.....University Club bldg., Z3 ALR
Howard, Harvey J.....Park Plaza Hotel, Z8 OP
James, William M.....508 N. Grand av., Z3 OP
Jones, Vincent L.....Missouri Theatre bldg., Z3 OALR
Kelley, Isaac D., Jr.....3720 Washington blvd., Z8 ALR
Kennedy, Francis L. .....Missouri Theatre bldg., Z3 ALR
Lange, Adolph Charles.....3903 Olive, Z8 OP
Leavy, C. A.....Metropolitan bldg., Z3 ALR
Leich, Charles F., Lt. (M.C.), U.S.N.R., Office of Naval
Officer Procurement, Missouri Pacific bldg. Z3 ALR
Luedde, W. H.....Metropolitan bldg., Z3 OP
Lyman, Harry W.....308 N. 6th, Z1 ALR
McMahon, Bernard John.....Missouri Theatre bldg., Z3 ALR
Mason, Roy E.....Frisco bldg., Z1 OP
Mattis, Robert Dean, Capt. (M.C.),
375 N. Taylor Avenue, Z8 OP
Meinberg, William H.....906 Olive, Z1 OP
Milder, Benjamin.....501 Humboldt bldg., Z3 OP
Miller, H. Edward.....634 N. Grand blvd., Z3 ALR
Milligan, Roy H.....3720 Washington blvd., Z8 ALR
Morman, William Daniel.....3507 Franklin av., Z6 ALR
Myers, E. Lee.....3903 Olive st., Z8 ALR
Nemours, Paul Roland.....1506 Hodiamont av., Z12 ALR
Payne, R. J.....University Club bldg., Z3 ALR
Pelz, Mort D.....Maj. (M.C.), 556 Purdue, University City ALR
Post, Lawrence T.....Metropolitan bldg., Z3 OP
Post, M. Hayward.....Metropolitan bldg., Z3 OP
Potter, Allen B.....Pasteur Medical bldg., Z8 ALR
Presnell, C. A.....634 N. Grand av., Z3 ALR

MISSOURI — Continued

Proetz, A. W.....Beaumont bldg., Z8 ALR
Reim, Hugo.....Metropolitan bldg., Z3 OP
Robertson, E. Norris, Jr., Washington University School of Medicine, 640 S. Kingshighway, Z10 OP..
Ryan, L. M.....Wall bldg., Z8 ALR
St. John, Dewey.....Metropolitan bldg., Z3 ALR
Sanders, Theodore E.....Metropolitan bldg., Z3 OP
Sauer, W. E.....Beaumont Medical bldg., Z8 ALR
Scholz, R. P.....Metropolitan bldg., Z3 ALR
Schwartz, Frederick O.....508 N. Grand blvd., Z3 OP
Shahan, W. E.....Metropolitan bldg., Z3 OP
Simpson, Robert K.....Col. (M.C.), 455 Lake av., Z8 OP
Smit, William M.....5285 Washington blvd., Z8 ALR
Stutsman, Albert C.....539 N. Grand blvd., Z3 ALR
Tooker, C. W.....Carleton bldg., Z1 OP
Walsh, Theodore E.....640 S. Kingshighway blvd., Z10 ALR
Webb, M. A.....4500 Olive st., Z8 ALR
Westlake, Samuel B.....Beaumont Medical bldg., Z8 ALR
Williams, John W., Capt. (M.C.), General Dispensary, 7th Service Command, 18th Floor, Continental bldg., Z8 ALR
Wood, V. Visscher.....Beaumont Medical bldg., Z8 ALR
Woodruff, Frederick E.....Metropolitan bldg., Z3 OP

SEDALIA

Brady, Charles Henry.....205 Ilgenfiety bldg. ALR

SPRINGFIELD

Bailey, Harold.....Landers bldg. OALR
Burch, Edward P. Maj. (M.C.), O'Reilly General Hospital OP
Cheek, Carey.....Medical Arts bldg. OP
Hauser, I. Jerome Maj. (M.C.), O'Reilly General Hospital ALR
Klingner, Thomas O.....Medical Arts bldg. OALR
Parry, Thayer L...Maj. (M.C.), O'Reilly General Hosp. OALR
Smith, Carlie Souter.....Holland bldg. OALR
Swift, Donald E.....Capt. (M.C.), O'Reilly Gen. Hosp. OP
Work, Walter P.....Capt. (M.C.), O'Reilly Gen. Hosp. ALR

TRENTON

Ambrose, Elmer C.....910 Main OALR

WEBSTER GROVES

Dyer, Clyde P.....Garelock bldg. OP

MONTANA

ANACONDA

Dunlap, Lawrence G.....101 Main OALR

BILLINGS

Morledge, Roy V.....Hart-Albin bldg. ALR
Morrison, Wm. R.....Hart-Albin bldg. OALR

GREAT FALLS

Hurd, Fritz D.....Medical Arts bldg. ALR
Lasky, Mortimer A., Maj. (M.C.), Base Hospital A.T.C., Army Air Base OP

MILES CITY

Rowen, Ernest Henry.....Box 570 ALR

MISSOULA

Marshall, William J.....Montana block ALR
Murphy, Edward S.....Col. (M.C.), 309 Eddy av. OP
Stephan, L. B., Capt. (M.C.).....438 Brooks st. OP

ROUNDUP

Lewis, George A.....213 Main OALR

NEBRASKA

COLUMBUS

Campbell, C. H.....2514½ Thirteenth OALR

CRETE

Peterson, John C., Lt. Comdr. (M.C.), Navy V-12 Doane College OP

FREMONT

Heine, Lyman H.....635 N. Main ALR
Heine, William Howard.....Odd Fellow bldg. ALR

GRAND ISLAND

Martin, Roy David.....Masonic bldg. ALR

KEARNEY

Wilcox, Malcolm B.....5 West 21st st. ALR

LINCOLN

Black, Paul.....Stuart bldg., Z8 OALR
Briggs, Henry Harrison, Jr., Major (M.C.), 3064 Puritan st., Z2 OP
Brooks, Earl B.....128 North 13th st., Z8 OALR
Cardwell, Edgar P., Major (M.C.), B.T.C. No. 6, Lincoln Field ALR
Hompes, J. J.....Sharp bldg., Z8 OALR
Patch, Orien B., Capt. (M.C.), U.S.A.A.F.T.T.C., Station Hospital, Lincoln Air Base, Z1 ALR
Sanderson, David D.....Stuart bldg., Z8 OALR
Thomas, James W.....Sharp bldg., Z8 OALR
Zemer, S. G.....Stuart bldg., Z8 OALR

McCOOK

Baum, William W., Major (M.C.), Army Air Base Hospital, Army Air Base OALR

NEBRASKA CITY

Stonecypher, D. D.....102½ S. 9th st. ALR

OMAHA

Bushman, L. B.....City National Bank bldg., Z2 OALR
Cassidy, W. A.....Medical Arts bldg., Z2 ALR
Cecha, Albin H.....4437 Woolworth av., Z5 OP
Coakley, Leo P.....Medical Arts bldg., Z2 ALR
Davis, John Calvin, Jr.....425 Aquila court, Z2 OALR
Finegan, J. F., Lt. (M.C.), USNR, 6241 Underwood, Z3 OP
Gifford, Harold.....Medical Arts bldg., Z2 OP
Hahn, William N.....City Nat'l Bank bldg., Z2 OP
Haney, William P.....Medical Arts bldg., Z2 OALR
Judd, J. H.....Medical Arts bldg., Z2 OP
Kully, Herman E.....Medical Arts bldg., Z2 ALR
Morrison, Wm. Howard.....Medical Arts bldg., Z2 OP
Potts, J. B.....3817 Dewey av., Z5 ALR
Rubendall, C.....Medical Arts bldg., Z2 OALR
Stokes, William H.....Medical Arts bldg., Z2 OP
Swab, Charles M.....Medical Arts bldg., Z2 OP
Watke, Frederic Martin.....Medical Arts bldg., Z2 ALR

NEVADA

LAS VEGAS

Richman, Frances.....750 Fremont st. OP

RENO

Clarke, Samuel T.....240 W. First st. OP
Creveling, Earle LaMont.....17 N. Virginia st. ALR
Fuller, John Andre.....First Nat'l Bank bldg. OALR
Shilling, Raymond L., Capt. (M.C.) Station Hospital, Army Air Base ALR
Strong, James C., Jr., Capt. (M.C.), Hq. Reno A.A.B. OP

NEW HAMPSHIRE

CONCORD
MacMillan, Andrew L., Jr.....46 Pleasant ALR

DOVER
Reid, Thomas F.....538 Central av. ALR

HANOVER
Burian, Hermann M.....The Eye Clinic OP
Murtagh, John Anthony.....Hitchcock Clinic ALR
Roper, Kenneth Lawrence
Clinical Division, Dartmouth Eye Institute OP

KEENE
Ford, LeRoy Sargent.....Lt. (M.C.), 30 School st. ALR

MANCHESTER
Monette, C. J.....814 Elm ALR
Rahmanop, Walter B.....1426 Elm ALR
Togus, Leopold T.....967 Elm ALR

NASHUA
Marcotte, Raymond H.....196 Main ALR
Nutter, C. F.....16 Amherst OP

PORTSMOUTH
Allen, Cary D.
Comdr. (M.C.), U. S. Naval Prison, Navy Yard ALR
Pollock, Frederic J.....Lt. (M.C.), Naval Hospital ALR

NEW JERSEY

ASBURY PARK
Baeseman, R. Winfield.....501 Grand av. OALR
Fisher, James A.....Jersey Central bldg. OALR
Upham, Helen F.....305 Third av. OALR

ATLANTIC CITY
Alfaro, V. R., Maj. (M.C.), England General Hospital ALR
Charlton, C. Coulter.....124 S. Illinois av. ALR
Howland, Alvin Wilcox..Maj. (M.C.), 3310 Fairmount av. OP
Pohl, Robert L., Maj. (M.C.),
Hq. A.A.F. Redistribution Center No. 1 ALR

CAMDEN
Goldstein, Henry Z., Capt. (M.C.),
AFIS Camden Induction Center ALR
Kline, Oram R.....514 Cooper ALR
Meyer, George P.....410 Haddon av. OP
Ruttenberg, Max.....303 Cooper ALR
Shemeley, William G., Jr.....7 Haddon av. OALR
Shipman, James S.....514 Cooper OP
Shope, Pierce .....511 Cooper OP

CAMP KILMER
Appelbaum, Alfred.....Maj. (M.C.), Station Hospital OP

CAPE MAY
Hunter, R. J., Lt. Comdr. (M.C.),
U.S.N.R., U. S. Naval Base ALR
Segal, Samuel, Jr.
Comdr. (M.C.), U. S. Naval Base Dispensary ALR

EAST ORANGE
Buvinger, Charles W.....50 Washington OALR
Campbell, William.....144 S. Harrison ALR
Cregar, John S.....150 Harrison OP

ELIZABETH
Ehrlich, Max.....Capt. (M.C.), 721 N. Broad, Z3 ALR
Gadomski, Casimir Francis
Lt. (M.C.), 331 S. Broad st., Z2 OP
Goldmacher, H. B.....113 Elmora av., Z2 ALR
Griesemer, Z. Lawrence.....1145 E. Jersey st., Z4 OP
Kapp, Carl G.....440 Westminster av., Z3 ALR
Sadoff, Irvin E.....Lt. (M.C.), 1045 North av., Z4 ALR

ENGLEWOOD
Worcester, George F.....220 Engle ALR

FORT DIX
Conley, John J...Capt. (M.C.), Tilton General Hospital ALR
Flanders, Merton N...Capt. (M.C.), Tilton General Hosp. ALR
Mason, Philip L...Capt. (M.C.), 145th Station Hosp. OP

GREYSTONE PARK
Gambill, Perry Judson.....New Jersey State Hospital ALR

FORT HANCOCK
Coleman, Joseph A.....Capt. (M.C.), Station Hospital ALR

HACKENSACK
Berke, Raynold N.....430 Union OP
Dezer, Charles N., Jr.....210 Main ALR
Greenfield, William John.....50 Anderson ALR
Hensle, Otto S.....Maj. (M.C.), 100 Prospect av. ALR
Liva, Arcangelo.....5 Pangborn place OALR
Spiegelglass, A. B.....417 Main ALR

HADDONFIELD
Shafer, A. H.....Maj. (M.C.), E. Greenman road ALR

JERSEY CITY
Pyle, Wallace.....15 Exchange place, Z2 OALR
Zbar, Joseph E.....90 Duncan av., Z6 ALR

LONG BRANCH
Campbell, W. K.....96 Third av. OALR
Strauss, Arthur.....130 Pavilion av. ALR

MAPLEWOOD
Sherman, Arthur E.....48 Headley Place OP-PI S

MONTCLAIR
Agnew, Hobart McV.....17 Plymouth OP
Shannon, James B.....66 S. Fullerton av. ALR
Shannon, L. M.....66 S. Fullerton av. ALR

MORRESTOWN
Ulmer, David H. B.....199 Chestnut ALR

MORRISTOWN
Byrne, J. Arthur.....16 Elm ALR
Mathews, Raymond H.....186 South ALR

NEWARK
Berney, Irving Vincent.....31 Lincoln Park, Z2 ALR
Eagleton, Wells P.....15 Lombardy, Z2 ALR
Hahn, William H.....15 Lombardy, Z2 OP
Hughes, L. W.....965 Broad, Z2 OP
Hurff, J. Wallace.....671 Broad st., Z2 OALR
Keim, William F.....25 Roseville av., Z7 ALR
Krone, William Frank.....31 Lincoln Park, Z2 ALR
O'Connor, D. F.....671 Broad, Z2 OALR
Orton, Henry B.....224 Delavan av., Z4 ALR

NEW JERSEY — Continued

Peer, L. A.....965 Broad, Z2 ALR-PI S
Rosen, Emanuel, Capt. (M.C.).....692 High, Z2 OP
Sherman, A. Russell.....671 Broad, Z2 OP
Sherman, Elbert S.....671 Broad, Z2 OP
Zehnder, A. Charles.....188 Roseville av., Z7 OP

NEW BRUNSWICK
Kler, Joseph Henry.....151 Livingston av. ALR

ORANGE
Emerson, Linn.....303 Park av. OP

PATERSON
Atwood, Edward A.....360 Park av. Z4 ALR

PERTH AMBOY
Cole, Nathan B.....Lt. Col. (M.C.), 104 Market OALR
Mann, J. J.....255 State ALR

PLAINFIELD
Childers, Robert Jefferson.....604 Park av. ALR
Levy, Abram.....1120 West Seventh st. OP

PRINCETON
Jellis, Walter, Lt. Comdr. (M.C.), Princeton University ALR

RIVERSIDE
Geary, Russell Diener.....337 Bridgeboro road ALR

SOUTH ORANGE
Adelman, Benjamin B., Lt. Comdr. (M.C.),
112 Wyoming av. OP
Miller, Joseph A.....364 Prospect st. ALR

SUMMIT
Burritt, Norman W.....30 Beechwood road ALR
Sly, John L.....382 Springfield av. ALR

TEANECK
Littwin, Charles.....950 Queen Anne road OALR

TRENTON
Ivins, William Clifford.....455 W. State, Z8 ALR
Levy, Irvin.....222 W. State st., Z8 OP

UNION CITY
Conty, Anthony J.....318 Forty-eighth ALR
Pagliughi, John Joseph.....401-18 st. ALR
Sacco, Anthony G.....2200 New York av. OALR

VENTNOR CITY
Dalton, Simon E.....101 S. Surrey av. ALR

WESTFIELD
Roh, Robert F., Lt. Comdr. (M.C.), USNR, 727 Glen av. ALR
Tomlinson, Rolland Davis.....505 East Broad st. ALR

WEST NEW YORK
Selinger, Samuel.....413 Sixtieth OALR

NEW MEXICO

ALAMOGORDO
Thorburn, O. L.....Maj. (M.C.) Station Hosp., A.A.B. ALR

ALBUQUERQUE
Brehmer, Harrison L.....First Nat'l Bank bldg. OALR

ROSWELL
Forrester, Alex M., Capt. (M.C.),
Roswell Army Air Field, Station Hosp. ALR

SANTA FE
Barton, William C.....104½ W. San Francisco OALR
Pumphrey, Gordon H.
Maj. (M.C.), Bruns General Hospital OP

NEW YORK

ALBANY
Bedell, Arthur J.....344 State, Z6 OP
Dowling J. Ivimey.....116 Washington av., Z6 OALR
Dubins, Henry B.....248 State st., Z6 OP
Freund, E. M.....762 Madison av., Z3 ALR
Holohan, Joseph Leo.....330 State, Z6 ALR
Judge, Harry V.....46 Willett, Z6 OP
Kemp, Roy C.....343 State, Z6 ALR
Mott, Walter Charles..Maj. (M.C.), 103 Lenox av., Z3 OP
Tebbutt, H. K.....240 State, Z6 ALR
Volk, Benjamin M.....146 Washington av., Z6 ALR

AUBURN
Bullard, C. W.....120 Genesee ALR
Wiley, Jason L.....Metcalf bldg. OP

BATAVIA
Bonner, Wm. F.....Veterans Administration Facility OALR
Wilson, Robert G.....304 East Main st. ALR

BATH
Adamski, Michael S.....8 Allen OP

BAY SHORE
McGrath, Harry.....12 Shore Lane OP

BINGHAMTON
Levy, Harry H.....119 Murray OP
Monserrate, Manuel Maria.....73 Main ALR
Randall, Hiram.....81 Main ALR
Smith, Windsor R.....67 Front ALR
Vogt, George C.....140 Chapin ALR
Watson, H. de Witt.....151 Front OALR

BRONXVILLE
Carter, William W.....6 Chestnut av. ALR
McKeown, Hugh S.....9 Tanglewylde av. OP
Petruzzi, William Astor.....65 Pondfield rd., Z8 ALR
Ross, John.....37 Oriole av. ALR

BROOKLYN
Agan, William B.....1 Nevins, Z17 OP
Baker, John Parcy.....51 S. Oxford, Z17 ALR
Behrens, Alvin.....9 Prospect Park West, Z15 OP
Beinfield, Henry H.....760 Eastern Parkway, Z13 ALR
Bergmann, Milton B.....959 Bushwick av., Z21 OP
Blankfield, Nathan.....429 Lincoln pl., Station B, Z16 ALR
Blumenfeld, Louis.....221 Brooklyn av., Z13 ALR
Brooks, Morris J.....455 Ocean av., Z26 ALR
Bruno, Joseph M. L.....31 Jefferson, Z6 OP
Buonaguro, Michael J.....589 Lorimer, Z11 OP
Campbell, G. S.
Capt. (M.C.), U. S. Naval Medical Supply Depot, Z1 ALR
Cowen, Jack P., Lt. (M.C.),
U. S. Naval Med. Supply Depot, Z1 OP
Cutino, R. M.....79 Pierrepont, Z2 OP
Cutler, Abraham Maurice.....750 Ocean av., Z26 ALR

NEW YORK — Continued

Ebeling, William B.....54 Greene av., Z5 OP
Evans, J. N.....23 Schermerhorn, Z2 OP
Feldman, Joseph.....668 Eastern Parkway, Z13 ALR
Feldman, Louis A.....259 New York av., Z26 OP
Fine, Abraham.....925 Ocean av., Z26 OALR
Fisher, George Garthwaite.....271 New York av., Z16 ALR
Freiman, George.....99 Lafayette av., Z17 OP
Garofalo, Charles Joseph.....439 Seventy-seventh, Z9 ALR
Gerzog, Bennett G.....891 St. Marks av., Z13 ALR
Gilligan, Alexander.....1154 Dean, Z16 ALR
Golden, Matthew G.....1 Nevins, Z17 ALR
Golding, Joseph E.....1 Nevins, Z17 OP
Gottlieb, Bernard N.....387 Eastern Parkway, Z16 ALR
Greenfield, Samuel D.....169 New York av., Z16 ALR
Griffin, Edwin A.....18 Prospect Park, West, Z15 ALR
Gubner, Julius.....25 Lefferts av., Z25 ALR
Hargitt, Charles A.....17 Schermerhorn, Z2 OP
Harris, Morris L.....20 Plaza st., Z17 ALR
Holzberger, John C.....7005 Freshpond, Z27 ALR
Hull, Allen.....401 76th st., Z9 OP
Kaplan, Carl.....69-17 Forest av., Z27 ALR
Kasnetz, Joseph P.....678 Eastern Parkway, Z13 ALR
Kaufman, Maxwell.....85 Maujer, Z6 ALR
Kayser, Reuben....442 Bay Ridge Parkway, 75th st., Z9 ALR
Keil, Peter A.....118 Pennsylvania av., Z7 ALR
Kopp, Mortimer M.....79 Argyle road, Z18 ALR
Kravitz, Daniel.....861 Park place, Z16 OP
Kraeg, Karl A.....167 Joralemon, Z2 ALR
Kruskal, I. D.....101 Lafayette av., Z17 OP
Kuritsky, Isadore.....1480 Nostrand av., Z26 ALR
Lloyd, Ralph I.....14 Eighth av., Z17 OP
Lombardo, Melchiore.....142 Joralemon st, Z2 OP
Mallon, Frank E.....1135 Park place, Z13 OP
Menger, Louis Charles.....202 Weirfield, Z21 ALR
Mensher, Ira Wadsworth.....50 Plaza st., Z17 OP
Mezz, David.....330 New York av., Z13 ALR
Moehle, Walter.....15 Schermerhorn, Z2 OP
Moore, Walter V.....1 Nevins, Z17 OP
Moorhead, Robert L.....125 Remsen, Z2 ALR
Nelson, Nathaniel F.....4910 15th av., Z19 OP
Neuberger, Julius Frederick
Capt. (M.C.), U. S. Naval Hospital ALR
Nova, Jules M.....349 Adelphi, Z5 OP
O'Brien, G. R., Comdr. (M.C.),
307 Sterling pl., Z17 ALR-PI S
Ohly, John H.....22 Schermerhorn, Z2 OP
Place, E. Clifford.....59 Livingston, Z2 OP
Rabbiner, Max....Maj. (M.C.), 358 New York av., Z13 ALR
Rizzuti, A. Benedict.....160 Henry st., Z2 OP
Rosenthal, Benjamin Charles....140 New York av., Z16 OP
Ruchman, Jacob.....301 Brooklyn av., Z13 ALR
Saskin, Edward.....855 St. Marks av., Z13 OP
Schmidt, James M.....40 S. Oxford, Z17 ALR
Scurti, Bernard V.....51 Harmon st., Z21 ALR
Sher, Maurice A.....5022 Fifteenth av., Z19 ALR
Shorr, Abraham.....225 Eastern Parkway, Z16 ALR
Silberman, Meyer.....266 Brooklyn av., Z13 ALR
Silverberg, Joseph S.....755 Ocean av., Z26 ALR
Sparber, Herman.....4701 Fifteenth av., Z19 OP
Steinbugler, William F. C.....515 Ocean av., Z26 OP
Stern, Wilhelm E.....1099 Bushwick av., Z21 ALR
Sunde, Einar A.....627 Second, Z15 ALR
Thaler, Nathan.....570 Eastern Parkway, Z25 ALR
Tucker, Frank Landale, Jr.....464 Ninth st., Z15 ALR
Usen, Louis.....278 New York av., Z16 ALR
Wechsler, A. H.....340 New York av., Z13 ALR
Weeth, Charles R.....37 Remsen, Z2 ALR
Weinstein, Samuel.....441 Ocean av., Z26 ALR
Welt, Bernard.....135 New York av., Z16 ALR
Wieselthier, Maurice L.....1322 Union st., Z13 OP
Zwerling, Samuel.....789 St. Mark's av., Z13 ALR

BUFFALO

Beyer, Louis J.....449 Delaware av., Z2 ALR
Bozer, Hermann Eugene.....468 Delaware av., Z2 ALR
Brown, C. M.....510 Delaware av., Z2 ALR
Buffum, Edward S.....40 W. North, Z2 ALR
Cott, C. C.....333 Linwood av., Z9 ALR
Cowper, H. W.....543 Franklin, Z2 OP
Fairbairn, John F.....925 Delaware av., Z9 ALR
Fowler, James Gwynne.....412 Linwood av., Z9 OP
Freeman, Sheldon B.....196 Linwood av., Z9 OP
Frost, Carl Grover.....333 Linwood av., Z9 ALR
Gerstner, Martin L.....468 Delaware av., Z2 ALR
Glosser, Herbert H.....448 Franklin, Z2 OP
Hague, Eliott B.....454 Franklin, Z2 OP
Harnick, Israel.....1132 Delaware av., Z9 ALR
Howard, Wm. M.....389 Linwood av., Z9 OP
Jones, W. Yerby.....436 Sycamore st., Z4 OP
King, Walter F.....519 Delaware av., Z2 OP
Koenig, Ivan J.....40 W. North, Z2 OP
Krystaf, Joseph George.....1004 Fillmore av., Z11 ALR
Le Win, Thurber.....112 Linwood av., Z9 OP
Luhr, A. F.....40 W. North, Z2 OP
McGregor, D'Arcy .....83 Bryant st., Z9 ALR
March, Clara A.....465 Ashland, Z13 OP
Palmer, Milton A.....2186 Seneca, Z10 OP
Rosenberg, Joseph.....333 Linwood av., Z9 ALR
Scanio, Joseph Charles.....333 Linwood av., Z9 ALR
Schutz, Joseph A.....2804 Main, Z14 OP
Smith, Leon.....467 Linwood av., Z9 ALR
Trotter, Homer A.....40 W. North, Z2 ALR
Wurtz, W. J. M.....333 Linwood av., Z9 ALR

CAMP SHANKS

Krall, J. Thomas.....Maj. (M.C.), Station Hosp. OP
Wegner, E. A., Jr., Capt. (M.C.).....Station Hospital ALR

CORNING

Armentrout, Lewis H., Jr.....22 E. 1st st. ALR

CORTLAND

Higgins, R. P.....20 Court OALR

CRAIGSVILLE

Overton, Jesse W.....Lt. (M.C.), USNR, Box 1 ALR

EAST AURORA

Andrews, H. D.....429 Oakland av. OP

ELMIRA

Boland, William Tilden.....315 Lake OP
Glover, Arthur C.....311 W. Church OALR

ELMONT, L. I.

Firestone, Charles.....225 Meachann av. ALR

FAR ROCKAWAY

Hirschmann, Hans A.....1502 Mott av. ALR

FLUSHING

Goode, Edward Allen.....143-28 Sanford av. ALR
Weiss, Harry.....Capt. (M.C.), 140-50 Ash Avenue OP

NEW YORK — Continued

FOREST HILLS L. I.
Kafka, M. Martyn.....109-05 72nd av. ALR

FORT TOTTEN
Vadala, Anthony J.....Lt. Col. (M.C.), Station Hospital OALR

FORT H. G. WRIGHT
Rothberg, Maurice.....Maj. (M.C.), Station Hospital OP

FREEPORT, L. I.
Rossman, Morris Muncie
Lt. (M.C.), U.S.N.R., 115 N. Main ALR

GASPORT
Pierce, Wm. W.....Maj. (M.C.) ALR

GENEVA
Spengler, John Arthur.....423 S. Main OP

GLEN FALLS
Buck, Clair A.....Maj. (M.C.), 191 Glen ALR

HEMPSTEAD
Austin, M. F.....Maj. (M.C.), 231 Main st. ALR
Coon, Eugene H.....131 Fulton av. ALR
Hughes, Wendell L.....Professional bldg. OP-PI S
Jarrott, James T.....131 Fulton av. OP
Maier, Marcus M.....513 Front st. ALR
Smith, Henry Buel.....131 Fulton av. ALR

HOLLIS
Bunnell, C. W.....Lt. Comdr. (M.C.), 9115 193rd st. ALR

HUNTINGTON
Kimbrig, Max.....20 High ALR

ITHACA
Wilson, Hudson J.....First Nat'l Bank bldg. ALR

JAMAICA, L. I.
Bender, M. S.....88-05 150th, Z2 ALR
Douglas, Edward M.....87-85 153rd, Z2 OP
Kane, J. T.....88-49 163rd, Z3 ALR
Keeler, Lansing H.....89-04 148th, Z2 OALR
Loewe, Walter R., Comdr. (M.C.), 180-27 Tudor rd., Z3 OP
Miller, Saul.....88-05 150th, Z2 OP
Minsky, Arthur.....8601 Homelawn st., Z3 OP

JAMESTOWN
Bourne, Malcolm G.....110 W. 3rd OP

JOHNSON CITY
Hudock, John Paul.....67 Broad st. ALR

JOHNSTOWN
Sarno, J. Frederick.....109 S. William st. ALR

KINGSTON
Wilson, Harold A.....71 Maiden Lane ALR

LANCASTER
Palmer, Milton Alwood.....18 Park blvd. OP

LITTLE FALLS
Newton, Morris H.....Burrell bldg. OP

LOCKPORT
Morgana, Dante James.....13 Main OP
Parker, Harry L.....88 East av. ALR

LONG ISLAND CITY
Gewanter, Robert.....41-44 44th st., Z4 ALR
Hulka, J. H.....30-28 36th st. OALR

MIDDLETOWN
Hicks, William J.....15 King ALR
Neller, Walter I.....121 Wickham av. OP

MILLERTON
Tabor, Gilbert Stevens.....11 Park Row ALR

MITCHELL FIELD, L. I.
Hannon, R. Emmet.....Capt. (M.C.), Station Hospital OP
Weymuller, Ernest A.....Capt. (M.C.), Station Hosp. ALR

MT. KISCO
Newlands, W. A.....359 East Main ALR

MT. VERNON
Houlahan, James M.....117 S. Second av. OP
Keefe, William John.....205 E. Sidney av. OALR
Morrone, Francis X.....117 South 2nd av. ALR
Yavelow, Charles S.....244 Bedford av. ALR

NEWBURGH
Browning, Birt Lee.....208 Grand ALR
McWilliam, Charles S.....Maj. (M.C.), 205 Liberty st. OP

NEW ROCHELLE
Beck, A. L.....421 Huguenot OALR
Gibson, William J.....650 Main st. ALR
Hetrick, L. E.....260 North av. ALR
Smith, Morley T.....421 Huguenot ALR

NEW YORK CITY
Aebli, Rudolph.....30 E. 40th, Z16 OP
Allan, William B.....410 Park av., Z22 OALR
Atkinson, Miles.....123 E. 61st, Z21 ALR
Auerbach, Julius.....897 Park av., Z21 ALR
Ballin, M. J.....25 E. 77st., Z21 ALR
Barnes, Mark Hopkins.....324 E. 41st st., Z17 OALR
Bassett, David I.....1075 Grand Concourse, Z52 ALR
Bender, M. S.....121 E. 60th, Z22 ALR
Berens, Conrad.....35 E. 70th, Z21 OP-PI S.
Berger, Isidore.....888 Grand Concourse, Bronx, Z51 ALR
Berliner, M. L.....57 W. 57th, Z19 OP
Bilchick, Edwin B.....Maj. (M.C.), 216 W. 89th, Z24 ALR
Bogart, Donald Weeks.....20 E. 53rd, Z22 OP
Bonaccolto, Girolamo.....654 Madison av., Z21 OP
Bowers, W. C.....17 E. 61st., Z21 ALR
Boyes, T. L.....654 Madison av., Z21 OP
Branigan, C. Hugh.....114 E. 54th st., Z22 OP
Breuning, Paul H.....133 E. 58th, Z22 ALR
Brighton, George R.....580 Park av., Z21 ALR
Broones, Otto.....302 W. 87th, Z24 ALR
Bruder, Joseph.....57 W. 57th, Z19 OALR
Buchbinder, Murray W., Maj. (M.C.), Armed Forces
Induction Station, 480 Lexington av. ALR
Burman, Herman J.....1235 Grand Concourse, Z52 ALR
Carroll, Frank D.....Eye Institute, 635 W. 165th, Z32 OP
Castroviejo, Ramon.....635 W. 165th, Z32 OP-PI S
Cinelli, Albert A.....1021 Park av., Z28 ALR
Cohen, Martin.....29 E. 64th, Z21 OP
Coleman, Lester Laudy.....162 E. 80th, Z21 ALR

NEW YORK — Continued

Conboy, John E.....2265 University av., Z53 OP
Constantine, Elizabeth Frederick....340 E. 63rd st., Z21 OP
Corya, Harold W.....30 E. 40th, Z16 ALR
Creasy, Raymond C.....67 E. 59th, Z22 ALR
Crespo, Jose E.....28 West 95th st., Z25 ALR
Cunning, Daniel S.....121 E. 60th Z22 OALR
Daley, Jacob.....104 E. 40th, Z16 ALR
Davidson, Morris.....16 Park av., Z16 OP
Davis, G. E.....14 E. 90th st., Z28 OALR
Delman, A. H., Maj. (M.C.), Veterans Administration Hospital, 130 W. Kingsbridge Road, Z63 ALR
Depping, Charles William.....17 W. 54th, Z19 ALR
Dewey, C. H., Lt. Col. (M.C.).....90 Church st., Z7 OALR
Dillemuth, Frederick Joseph.....404 E. 154th, Z55 ALR
Dixon, G. S.....255 W. 108th, Z25 R
Dlugasch, Louis.....110 E. 36th, Z16 ALR
Doherty, W. B.....150 W. 55th, Z19 OP
Druss, Joseph George.....1040 Park av., Z28 ALR
Dunnington, J. H.....30 W. 59th, Z19 OP-PI S
Dwyer, James G.....375 Park av., Z22 ALR
Esterman, Benjamin.....983 Park av., Z28 OP
Everett, A. Reginald.....11 Wall st., Z16 ALR
Falkson, Kurt F.....229 W. 78th st., Z24 ALR
Fernlund, George Byron 8620 115th st., Richmond Hill, Z18 ALR
Fitzgerald, Fred J. C.....118 E. 65th, Z21 OP
Flack, J. Vincent....Lt. Comdr. (M.C.), 90 Church, Z7 OP
Foster, Harold.....92-11 35th av., Jackson Heights ALR
Fowler, Robert H.....610 Park av., Z21 ALR
Fowlkes, John Winston.....12 E. 63rd, Z21 ALR
Francis, Charles C.....610 West End av., Z24 ALR
Fread, Bernard.....45 E. 85th, Z28 OP
Frey, W. G., Jr.....121 E. 60th, Z22 OP
Friedman, Benjamin, Lt. Comdr. (M.C.), 201 W. 89th st. OP
Friesner, Isidore.....101 E. 73rd, Z21 A
Frothingham, Florie D.....133 E. 58th, Z22 ALR
Garrick, Thomas J.....40 E. 61st, Z21 ALR
Gartner, Samuel.....1749 Grand Concourse, Z53 OP
Gatewood, Wm. L.....111 E. 61st st., Z21 ALR
Gilmore, Goodlatte Browne..2940 Grand Concourse, Z58 ALR
Givner, Isadore.....108 E. 66th, Z21 OP
Glushak, Leopold.....1 West 85th, Z19 ALR
Goldman, H. G.....57 W. 58th, Z19 ALR
Goldman, Irving Brice.....121 E. 60th, Z22 ALR
Gould, Nathaniel Jay.....940 Grand Concourse, Z56 OP
Graebner, Herbert.....45 Park av., Z16 ALR
Graef, Fred W.....140 East 54th St., Z22 ALR
Grant, Maurice David.....1860 Morris av., Z53 ALR
Greene, James S.....61 Irving place, Z3 ALR
Greenfield, William J.....30 E. 40th av., Z16 ALR
Gresser, Edward Bellamy.....39 E. 75th, Z21 OP
Griesman, Bruno L.....1100 Park av., Z28 ALR
Griffey, Walter P.....Marine Hospital, Stapleton OP
Grimaldi, Americo John.....100 W. 59th, Z19 OP
Grove, Russell Clark.....60 E. 58th, Z22 ALR
Gurtov, John Jacob.....1235 Grand Concourse, Z52 ALR
Guy, Loren P.....40 E. 62nd, Z21 OP
Hanley, James Swift.....40 W. 55th, Z19 ALR
Hardy, LeGrand H.....23 E. 79th, Z21 OP
Hartshorne, Isaac.....30 W. 59th, Z19 OP
Haskin, W. H.....375 Park av., Z22 ALR
Herzig, Arthur J.....667 Madison av., Z21 ALR
Hetrick, J. A. Werner.....667 Madison av., Z21 ALR
Hetrick, L. E.....40 E. 61st, Z21 ALR
Holden, William H.....3 E. 85th, Z28 ALR
Hory, Joseph S.....2488 Grand Concourse, Z58 OP
Hunt, Westley Marshall.....907 5th av., Z21 ALR
Hynes, Frank Joseph.....121 E. 60th, Z22 ALR
Ide, David.....1749 Grand Concourse, Z53 ALR
Imperatori, C. J.....108 E. 38th, Z16 ALR
Ingalls, Raymond George.....73 East 71 st., Z21 OP
Jacobius, Lawrence.....2021 Grand Concourse, Z53 ALR
Jacobs, S. M.....15 West 81st st., Z24 OALR
Jaffe, Morris.....940 Grand Concourse, Z56 OP
Jessel, Erwin.....Capt. (M.C.), 204 W. 14th st. OP
Johnson, Thomas Holland.....30 W. 59th, Z19 OP
Jones, David H.....140 E. 54th, Z22 LR
Jones, Marvin F.....121 E. 60th, Z22 ALR
Judd, H. B.....40 E. 61st, Z21 ALR
Keil, Frank C.....660 Madison av., Z21 OALR
Kelly, Joseph Dominic.....140 E. 54th, Z22 OALR-PI S
Kend, Lee.....133 E. 58th, Z22 ALR
Kenyon, B. B., Lt. Comdr. (M.C.), Fort Schuyler, Bronx, Z61 ALR
Kirby, D. B.....780 Park av., Z21 OP-PI S
Kirkbright, James C.....40 E. 61st, Z21 ALR
Kleinfeld, Louis.....17 E. 84th, Z28 ALR
Knapp, Arnold.....10 E. 54th, Z22 OP
Knighton, W. S.....121 E. 61st, Z21 OP
Knopf, Saul.....30 E. 40th st., Z16 ALR
Koch, Ferdinand L. P.....780 Park av., Z21 OP
Koller, Carl.....68 E. 86th, Z28 OP
Kopetzky, S. J.....Col. (M.C.), 71 E. 80th st., Z21 ALR
Kornberg, Maurice.....441 E. 15th, Z3 ALR
Kornzweig, Abraham L.....1200 Fifth av., Z29 OP
Kronenberg, Bernard.....162 W. 54th, Z19 OP
Krug, Ernest F.....988 Fifth av., Z21 OP
LaGattula, Frank.....2488 Grand Concourse, Bronx 58 OP
Lambert, Robert K.....1010 Fifth av., Z28 OP
Last, Murray A.....1095 Park av., Z28 OP
Laval, Joseph.....136 E. 64th, Z21 OP
La Vigne, A. A.....667 Madison av., Z21 OALR
Law, Frederick M.....140 E. 54th, Z22 ALR
Lefkon, Irving M.....37-14 73rd, Jackson Heights ALR
Lempert, Julius.....119 E. 74th st., Z21 ALR
Levbarg, John J., Maj. (M.C.)....176 W. 87 st., Z24 ALR
Liggett, Harold.....133 E. 58th, Z22 ALR
Limbach, Earl Frederick..30 W. 59th, Station "G", Z19 ALR
Lore, John M.....325 West End av., Z23 ALR
McAuliffe, G. W.....630 Park av., Z21 ALR-PI S
McClanahan, Robert.....60 E. 75th, Z21 OP
McCuaig, Victor C.....40 E. 61st, Z21 ALR
McDannald, C. E.....100 W. 59th, Z19 OP
McGavic, John Samuel.....635 W. 165th, Z32 OP
McGrath, James B.....2615 Grand Concourse, Z58 ALR
McKeown, Hugh S.....635 W. 165th, Z32 OP
McLean, John M.....525 E. 68th, Z21 OP
Macdonald, James O.....67 E. 59th st., Z22 ALR
MacDonald, Philip G.....115 E. 61st st.,Z21 ALR
Macpherson, Duncan.....920 Fifth av., Z21 ALR
Maliniac, Jacques W.....106 E. 78th, Z21 ALR-PI S
Martin, Ashby G.....895 Lexington av., Z21 ALR
Matusow, Abraham P.....1100 Grand Concourse, Z56 ALR
Maybaum, J. L.....1070 Park av., Z28 ALR
Miller, Isidore.....2021 Grand Concourse, Z53 ALR
Miller, Joseph William.....242 E. 19th, Z3 ALR
Minsky, Arthur.....2 E. 95th st., Z28 OP
Minsky, Henry.....2 E. 95th, Z28 OP
Mirow, David.....237 E. 20th, Z3 ALR
Mittendorf, A. D.....399 Park av., Z22 OP
Moshinsky, Alexander L.....237 E. 20th, Z3 ALR

NEW YORK — Continued

Moyer, John S.....410 Park av., Z22 ALR
Myerson, M. C.....136 E. 64th, Z21 ALR
Neer, Edmonde DeWitte.....115 E. 61st, Z21 ALR
Neuschatz, Gerald.....327 Central Park West, Z25 ALR
Nisselson, Max.....250 Riverside drive, Z25 ALR
Northcott, Thomas A.....40 E. 61st, Z21 OP
O'Kane, George H., Lt. Comdr. (M.C.),
Office of Naval Procurement, 33 Pine st. ALR
Opin, Edward.....2065 Grand Concourse, Z53 ALR
Page, John R.....127 E. 62nd, Z21 ALR
Palmer, Arthur.....667 Madison av., Z21 ALR-PI S
Paton, R. T.....927 Park av., Z28 OP
Pearlman, Louis M.....53 E. 93rd, Z28 ALR
Perera, Charles A.....70 E. 66th, Z21 OP
Perkins, Osborn P.....121 E. 60th, Z22 OP
Pfeiffer, Raymond L.....635 W. 165th, Z32 OP
Pullen, Edward Markey.....30 E. 40th, Z16 ALR
Reese, Algernon B.....73 E. 71st, Z21 OP
Risch, Otto C.....104 E. 40th, Z16 ALR
Rolett, D. M.....876 Park av., Z21 OP
Rosen, David M.....19 W. 55 st., Z19 ALR
Rosen, Sam.....101 E. 73rd, Z21 ALR
Rosenbaum, Morris.....61 W. 88th, Z24 OP
Rosenbluff, Michael.....225 W. 106th, Z25 ALR
Rosenwasser, Harry.....101 E. 73rd st., Z21 ALR
Ross, John.....6 E. 70th, Z21 ALR
Rubenstein, Hyman John.....510 Madison av., Z22 ALR
Ryan, Maxwell D.....660 Madison av., Z21 ALR
Sackin, David..."St. Moritz," 50 Central Park South, Z19 ALR
Saltzman, Samuel L.....334 W. 86th, Z24 OP
Samuels, Bernard.....57 W. 57th, Z19 OP
Sauer, John J.....30 E. 40 st., Z16 OP
Saunders, Truman L.....103 E. 65th, Z21 ALR
Scal, Joseph Coleman.....10 E. 96th, Z28 ALR
Schechter, Solomon P.....1749 Grand Concourse, Z53 ALR
Scheer, Henry M.....522 West End av., Z24 ALR
Schillinger, Raphael.....320 West End av., Z23 ALR
Schlivek, Kaufman.....1016 Fifth av., Z28 OP
Schmidt, Otto V. M.....568 Park av., Z21 ALR
Schoenberg, Mark J.....667 Madison av., Z21 OP
Schugt, Henry P.....122 E. 76th, Z21 ALR
Schwartz, A. A.....7 W. 71st, Z23 ALR
Schwartz, Harry.....1749 Grand Concourse, Z53 OP
Schwartz, Leo.....136 E. 57th, Z22 ALR
Seward, J. A.....115 E. 61st, Z21 ALR
Sitchevska, Olga L.....30 Fifth av., Z11 OP
Smith, Clarence Henry.....140 E. 54th, Z22 ALR
Smith, James M.....123 E. 53rd, Z22 ALR
Smith, James W.....1016 Fifth av., Z28 OP
Sparer, William S.....50 E. 10th, Z3 ALR
Stark, Jessie B.....46 Park av., Z16 OP
Steinbach, Louis.....1882 Grand Concourse, Z57 OP
Stovin, Joseph S.....151 Central Park West, Z23 ALR
Straatsma, Clarence R., Lt. Comdr. (M.C.)
66 E. 79th, Z21 ALR-PI S
Street, Alvin Murray.....745 5th av., Z22 ALR
Strickler, Joseph George.....200 W. 57th, Z19 ALR
Sturges, Leigh F.....121 E. 60th, Z22 ALR
Swick, David A.....30 W. 70th, Z23 OP
Syracuse, Victor R.....29 Washington Sq. W., Z11 OP
Tickle, Thomas G.....30 E. 40th st., Z16 ALR
Troncoso, Manuel U.....500 West End av., Z24 OP
Tusak, E. A.....115 E. 90th, Z28 OP
Vogel, Felix P. S.....1749 Grand Concourse, Z53 ALR
Watnick, Joseph Louis.....10 W. 96th, Z25 ALR
Webster, David H.....140 E. 54th, Z22 OP
Wexler, David.....103 E. 86th, Z28 OP
Wheeler, Maynard C.....30 W. 59th, Z19 OP
White, Francis W.....471 Park av., Z22 ALR
White, James W.....15 Park av., Z16 OP
Wiener, Alfred.....550 Park av., Z21 OALR
Wincor, Henry G.....1882 Grand Concourse, Z57 OP
Woodman, DeGraaf.....156 E. 37th, Z16 ALR
Zuckerman, Joshua.....875 Park av., Z21 OP

NIAGARA FALLS

Goetzman, Arthur C...Maj. (M.C.), 8907 Hennepin av. OP
Price, Norman W.
Hyde Park Blvd. & Lewiston Rd., Route No. 47 OALR
Rooker, A. M.....United bldg. ALR
Walker, Oliver Frank.....2873 Lewiston road ALR

NYACK

Theis, Franklyn B.....209 S. Broadway ALR
Toms, S. W. S.....120 S. Broadway OALR

OLEAN

Moore, Ellis Columbus.....Nat'l Bank bldg. ALR

ONEIDA

Crockett, R. L.....112 Farrier av. OP

ONEONTA

Hall, Edwin P.....76 Chestnut ALR

POUGHKEEPSIE

Kreiger, William A.....103 Hooker av. OALR
Miller, Sidney N.....113 Hooker av. ALR
Voorhees, Earle W.....8 Garfield place ALR

QUEENS VILLAGE

Pauley, Gerald E.....9460—220th st. ALR
Roemaet, Charles M.....9404 207 st., Z8 ALR

ROCHESTER

Avery, Elroy J.....11 N. Goodman, Z7 ALR
Barber F.....75 S. Fitzhugh, Z8 OP
Bishop, Vernon L.....277 Alexander, Z7 ALR
Clark, L. H.....337 Monroe av., Z7 OALR
Elliott, Raymond E.....76 S. Fitzhugh, Z8 ALR
Fischer, Abraham.....Mercantile bldg., Z4 OP
Gipner, J. F.....Medical Arts bldg., Z7 OP
Gorin, Meyer.....35 Chestnut st., Z4 ALR
Hawkins, Raymond W.....39 S. Goodman, Z7 ALR
Hilleman, George A.....267 Alexander, Z7 ALR
Honiss, John Ralph.....277 Alexander, Z7 ALR
Jones, Leonard W.....16 N. Goodman, Z7 OALR
Kennedy, Eldred Weston....Gas and Electric bldg., Z4 OP
Lerner, Macy Levi.....332 Park av., Z7 OP
McDowell, N. D.....275 Alexander, Z7 OALR
Morris, A. G.....Medical Arts bldg., Z7 ALR
Nash, C. Stewart.....277 Alexander, Z7 ALR
O'Brien, Elmer W.....5 N. Goodman, Z7 ALR
Shapero, I. M.....289 Alexander, Z7 OALR
Snell, Albert C.....53 S. Fitzhugh, Z4 OP
Sullivan, Charles T.....277 Alexander, Z7 OP

ROCKVILLE CENTER, L. I.

Donlon, John J.....21 S. Village av. ALR
Goldman, A. Milton.....1 N. Village av. OP
Preefer, Charles Joseph.....195 Hempstead av. OP

NEW YORK — Continued

ROME
Valone, Frank H.....107 W. Court ALR

RYE
Carroll, Frank D.....Blind Brook Lodge OP
Peterson, N. Vern.....Highland Hall ALR

ST. ALBANS, L. I.
Braley, A. E., Lt. Comdr. (M.C.),
USNR, U. S. Naval/Hospital, Z12 OP
Gerstley, Manfred J., Comdr. (M.C.),
U. S. Naval Hospital, Z12 ALR

SAMPSON
Livshin, Norman, Lt. Comdr. (M.C.), USNR,
U. S. Naval Training Station ALR
McLaughlin, John J.
Lt. (M.C.), U. S. Naval Training Station ALR

SARANAC LAKE
Beck, Robert J.....105 Main OP
Wilson, George E.....108 Main ALR

SARATOGA SPRINGS
Goodfellow, Thomas J.....Arcade bldg. OALR

SCARSDALE
Austen, Willard Emerson, Lt. Comdr. (M.C.),
Scarsdale Manor, Apt 4D North ALR
Ryan, E. P.....Lt. Comdr. (M.C.), 18 Fairview road OP

SCHENECTADY
Gulick, John Duncan.....704 Union, Z5 ALR
Ostrom, William C.....1350 Union, Z8 OP

STATEN ISLAND
Conway, William S.
Capt. (M.C.), Halloran General Hospital OALR
DeVoe, Arthur Gerard, Capt. (M.C.), Halloran Gen Hosp. OP
Kahn, K. M., Lt. Col. (M.C.), Halloran General Hospital ALR
Seligman, Albert E.....41 Forest av., Z1 ALR

SYRACUSE
Blaisdell, Iri Hazard.....Medical Arts bldg., Z2 ALR
Brown, Mortimer G.....State Tower bldg., Z2 ALR
Brust, H. O.....820 S. Crouse av., Z1 ALR
Cahill, James F.....428 S. Salina, Z2 OP
Cave, Linus S.....State Tower bldg., Z2 ALR
Dickinson, William G.....State Tower bldg., Z2 OP
Gillette, D. F.....State Tower bldg., Z2 OP
Hoople, Gordon D...Maj. (M.C.), Medical Arts bldg., Z2 ALR
Joy, Harold Henry.....State Tower bldg., Z2 OP
Kauffman, A. B.....Chimes bldg., Z2 ALR
Kline, H. G.....State Tower bldg., Z2 ALR
Moore, Donald E., Lt. Comdr. (M.C.), USN
1100 Euclid av., Z10 OP
Moore, Roy S.....State Tower bldg., Z2 ALR
O'Connor, Frederick J.....State Tower bldg., Z2 ALR
Rubenstein, A. H.....Medical Arts bldg., Z2 ALR
Webster, F. R.....State Tower bldg., Z2 OP

TARRYTOWN
Newlands, W. Alexander.....19 S. Broadway ALR

TROY
Sulzman, Frank Malcolm.....1831 Fifth av. OALR
Sulzman, John H..Lt. (M.C.), U.S.N.R., 1831 Fifth av. OALR

UTICA
Beattie, William H.....258 Genesee, Z2 OALR
Farrell, James I.....Lt. Comdr. (M.C.), 250 Genesee, Z2 OP
Gruppe, Karl.....258 Genesee st., Z2 ALR
Jones, Fred G.....258 Genesee, Z2 ALR

WARSAW
Nairn, George Waverly
Wyoming County Community Hospital ALR

WATERTOWN
Atkinson, John H.....168-70 Sterling ALR
Atkinson, Walter S.....129 Clinton OP
Henderson, L. E.....129 Clinton OP
McAskill, James E.....Woolworth bldg. ALR

WEST POINT
Martin, H. F.....Maj. (M.C.), Station Hospital ALR

WHITE PLAINS
Emery, John R.....Medical Centre ALR
Knight, Granville F.....Medical Centre bldg. ALR
Moriarty, Richard W.....3 Mamaroneck road OALR

WOODHAVEN, L. I.
Frothingham, Edward.....8626 94th st., Z21 OALR
Stak, James Edward.....8838 Woodhaven blvd., Z21 ALR

YONKERS
Fitzgerald, Wm. W.....27 Ludlow OP
Petti, George Hyacinth.....4 Berkeley av. ALR
Wolcott, Charles C.....27 Ludlow ALR

NORTH CAROLINA

ASHEVILLE
Chapman, E. J.....331 Haywood bldg. ALR
McGowan, Joseph Francis.....New Medical bldg. ALR
Swann, Cecil C.....Flat Iron bldg. ALR
Weizenblatt, Sprinza.....709 New Medical bldg. OP
Furman, Martin A., Maj. (M.C.),
Station Hospital, Camp Mackall ALR

CHARLOTTE
Johnston, James G.....1520 E. 4th, Z4 OP
Motley, Fred E.....106 W. 7th, Z2 ALR
Neblett, Herbert Clarence.....401 N. Tryon, Z2 OP
Peeler, C. N.....106 W. 7th, Z2 OALR
Sloan, Henry L.....106 W. 7th, Z2 OP
Smith, Franklin C.
Charlotte Eye, Ear and Throat Hospital, Z2 OP
Whisnant, A. M.....Professional bldg., Z2 OALR

CHERRY POINT
Northington, Page, Capt. (M.C.),
U. S. Marine Corps Air Station ALR
Snyder, Eugene Ralph, Lt. Comdr. (M.C.),
Marine Air Station, M.A.G. 34 ALR

DURHAM
Anderson, William Banks.....Duke Hospital OP
Eagle, Watt Weems.....Duke Hospital ALR
Ferguson, George Burton.....1111 West Main ALR
Kerns, Thomas C.....1110 W. Main ALR
McPherson, Samuel D.....McPherson Hospital ALR

FORT BRAGG
Arnold, Ralph A...Capt. (M.C.), 65th General Hospital ALR

NORTH CAROLINA — Continued

GOLDSBORO
Sheppard, Louis B., Maj. (M.C.), Seymour Johnson Field OP

GREENSBORO
Cohen, Jerome H., Capt. (M.C.), Station Hospital, B. T. C. No. 10 OP
Taylor, Shahane R.....Jefferson bldg. ALR
Wolfe, Hugh C.....102 N. Elm ALR

HENDERSON
White, Clarence Hunt.....Box 257 ALR

KINSTON
Peery, Vanca Price.....Kinston Clinic ALR

MORGANTON
Billings, Gilbert M.....110 S. Sterling ALR

NEW RIVER
Knowlton, Don S., Capt. (M.C.), Med. Field Service School School Battalion Training Center, Camp LeJeune ALR
Smoot, M. C., Lt. Comdr. (M.C.).....Camp LeJuene ALR

RALEIGH
Hicks, Vonnie Monroe.....Professional bldg. OP
Powers, Frank P.....Professional bldg. ALR

SALISBURY
McCutchan, Frank.....100 W. Innes ALR

SWANNANOA
Lathbury, Edwin, Maj. (M.C.), Moore General Hospital ALR

WINSTON-SALEM
Combs, Fielding.....Nissen bldg., Z3 ALR
Davis, Thomas W.....O'Hanlon bldg., Z3 OALR
Harrill, James Albert.....Bowman Gray School of Med. ALR
Sink, Vergil Rex.....852 W. 5th, Z5 ALR
Speas, William Paul.....Reynolds bldg., Z3 OP

NORTH DAKOTA

BISMARCK
Buckingham, Tracy W.....405 Broadway ALR
Constans, George Maurice.....Clinic bldg. OP
Diven, Wilbur Lester.....406½ Main ALR
Rosenberger, Henry P.....221 Fifth ALR

FARGO
Foster, George C.....Fargo Clinic OALR
Larson, G. Arthur.....A. O. U. W. bldg. OALR
Rindlaub, Elizabeth P.....321 Eighth South OALR
Rindlaub, John H.....321 Eighth South OALR
Winn, William R.....Dakota Clinic ALR

GRAND FORKS
Alger, L. James.....Box 328 ALR
Jensen, August Frithiof.....322 De Mers av. OP
Youngs, Nelson A.....Grand Forks Clinic ALR

MINOT
Brunner, George Harmon.....McCannel Clinic bldg. OP
Lampert, M. T.....Northwest Clinic ALR
McCannel, Archie D. Corner 1st st., S. W. and Central av. OALR

OHIO

AKRON
Arbuckle, David S.....Akron Savings & Loan bldg., Z8 OALR
Brumbaugh, John D., Capt. (M.C.), 017351 75 North Wheaton Road, Z13 OALR
Fowler, Floyd J.....Akron Clinic 513 W. Market st., Z3 ALR
Grimmett, Roger Skelton 406 Akron Savings & Loan Building, Z8 ALR
Mathias, Daniel F. 819 First Central Tower bldg., Z8 ALR
Springer, James E.....409 Peoples Bank bldg., Z8 ALR

ASHTABULA
O'Connor, Timothy Francis.....4634 Main av. ALR

BELLEFONTAINE
Butler, Robert H.....112 W. Columbus av. OALR
Harbert, John P.....135 N. Main OALR

CAMBRIDGE
Levitt, Jesse M., Capt. (M.C.), Fletcher General Hospital OP

CANTON
Hoffmann, John N.....Brant bldg., Z2 ALR
King, Clair Boyd.....First Nat'l Bank bldg., Z2 OP
Rosedale, Raymond Samuel....610 N. Market av., Z2 ALR
Schirack, Clarence J.....817 Cleveland av. N. W., Z3 ALR
Toot, Joseph F.....First Nat'l Bank bldg., Z2 OP
Underwood, John H.....Brant bldg., Z2 ALR
Weaver, Homer V.....625 Cleveland av. N. W., Z3 ALR

CHILLICOTHE
Shepard, Emerson P.....72 West Second ALR

CINCINNATI
Asbury, Mary K.....3549 Holly Lane, Z8 OP
Beyer, A. G.....Carew Tower, Z2 OALR
Brown, Albert Louis.....Carew Tower, Z2 OP
Finney, Nancy E.....2525 Gilbert av., Z6 OP
Fischbach, Victor W.....Union Central bldg., Z2 ALR
Goodyear, H. M.....Doctors bldg., Z2 ALR
Haggart, Harry H.....No. 1 Handasyde Lane, Z8 ALR
Hans, C. L.....Provident Bank bldg., Z2 OALR
Heidelman, George.....Union Central bldg., Z2 OP
Hendricks, Louis J.....Carew Tower, Z2 OP
Hickman, W. R.....Capt. (M.C.), Broadway Hotel ALR
Hofling, Charles A.....707 Race, Z2 OP
Hofmann, Albert P.....630 Vine, Z2 ALR
Howard, Robert E.....Union Central bldg., Z2 ALR
Hyman, Morris, Capt. (M.C.), 1139 Towanda Ter., Z16 ALR
Iglauer, Samuel.....707 Race, Z2 ALR-PI S
King, Edward.....Carew Tower, Z2 ALR
Levin, Abram Ascher.....Carew Tower, Z2 OP
Lyle, Donald J.....Carew Tower, Z2 OP
Miller, Ralph H., Capt. (M.C.), 6257 Rogers Park Place, Pleasant Ridge, Z13 OP
Mithoefer, W.....Carew Tower, Z2 ALR
Portnoy, Samuel H.....19 Garfield Place, Z2 ALR
Ray, Victor, Jr.....Carew Tower, Z2 OP
Reid, H. W.....Union Central bldg., Z2 OF
Safer, Louis A.....Provident Bank bldg., Z2 ALR
Sakler, Barnet R...Capt. (M.C.), 695 N. Crescent, Z29 OP
Schrimpf, Cyril Edward.....Union Central bldg., Z2 OP
Seltz, Samuel.....Doctors bldg., Z2 ALR
Siegel, Francis X.....Union Central bldg., Z2 OALR
Stanbery, Henry.....Doctors bldg., Z2 OP

OHIO — Continued

Stewart, T. M.....Union Central bldg., Z2 OP
Stitt, H. L.....Doctors bldg., Z2 ALR
Stoll, Karl Ludwig.....19 W. 7th, Z2 OP
Swing, Frank U.....Doctors bldg., Z2 ALR
Tangeman, H. F.....Union Central bldg., Z2 ALR
Thompson, E. H.....19 W. 7th, Z2 OALR
Woodward, Paul Maxwell.....4140 Hamilton av., Z23 ALR

CLEVELAND

Adelstein, Fred.....Rose bldg., Z15 ALR
Allen, Donald Gray.....1021 Prospect av., Z15 OP
Bruner, Abram Breneman.....Guardian bldg., Z14 OP
Bruner, William E.....Guardian bldg., Z14 OP
Bucher, Walter M.....4187 Pearl road, Z9 ALR
Burke, T. A.....Rose bldg., Z15 OALR
Chamberlain, Webb Parks.....7405 Detroit av., Z2 OP
Collins, James T.....Republic bldg., Z15 ALR
Deeton, William L.....13125 Shaker square, Z20 ALR
Dixon, Fred Willis.....Rose bldg., Z15 ALR
Engler, Clarence William.....2323 Prospect av., Z15 ALR
Forsythe, S. T.....Rose bldg., Z15 OALR
Garrett, Ernest W.....Hanna bldg., Z15 ALR
Hamilton, Howard Eugene.....10515 Carnegie av., Z6 ALR
Harris, Harold Earl.....Cleveland Clinic, Z6 ALR
Jackson, Clarence C. R.....Lakeside Hospital, Z6 ALR
Jacoby, Mark W.....Hanna bldg., Z15 OP
Johnson, Lorand V.....Lakeside Hospital, Z6 OP
Kennedy, Roscoe J., Lt. Comdr. (M.C.),
3342 Sutton Rd., Z20 OP
Kinney, Charles Ed.....10515 Carnegie av., Z6 ALR
Large, S. H.....170 Eddy rd. ALR
Lemel, Samuel L.....1030 Euclid av., Z15 ALR
McCall, J. W.....610 Rose bldg., Z15 ALR
McDonald, C. L.....Rose bldg., Z15 OALR
Metz, Roy B.....1422 Euclid av., Z15 OP
Mitchell, Horace E.....14805 Detroit av., Z7 ALR
Monson, S. H.....B. F. Keith bldg., Z15 OALR
Moore, Paul G.....Republic bldg., Z15 OP
Moore, Paul M., Jr.....Cleveland Clinic, Z6 ALR
Motto, M. Paul.....Rose bldg., Z15 OP
Mussun, W. G.....Rose bldg., Z15 ALR
Nelson, Charles F.....Schofield bldg., Z15 OALR
Phelan, Harold V.....Rose bldg., Z15 OP
Pitkin, C. E.....Carnegie Medical bldg., Z6 ALR
Quittner, S. S.....10515 Carnegie av., Z6 OALR
Rieger, W. H.....Hanna bldg., Z15 ALR
Rosenberger, Harry Clifford.....10515 Carnegie av., Z6 ALR
Ruedemann, Albert D.....Cleveland Clinic, Z6 OP
Ryall, George W.
17010 Kinsman road, Shaker Heights, Z20 ALR
Schwarz, Gerald T.....10515 Carnegie av., Z6 OP
Shiras, Howard H.....Guardian bldg., Z14 OP
Somberg, Joseph S.....406 Bulkley, Z15 OP
Stoffregen, Charles Henry.....10515 Carnegie av., Z6 ALR
Stotter, Arthur L.....1148 Euclid av., Z15 OALR
Thoburn, Thomas W.....1020 Huron road., Z15 ALR
Thomas, Charles I.....10515 Carnegie av., Z6 OP
Tuckerman, W. H.....1020 Prospect av., Z15 OALR
Weidlein, Ivan F.....10515 Carnegie av., Z6 ALR

COLUMBUS

Beatty, H. G.....150 E. Broad, Z15 ALR-PI S
Brown, John E.....370 E. Town, Z15 OALR
Clark, I. G.....188 E. State, Z15 OALR
Davies, Morgan C.....269 E. State, Z15 OP
Davis, William C.....150 E. Broad, Z15 OP
Emswiler, Herbert D.....370 E. Town, Z15 ALR
Frost, Albert Dalbey.....150 E. Broad, Z15 OP
Hauer, A. M.....327 E. State, Z15 ALR
Means, R. G.....40 S. Third, Z15 ALR
Odom, Stanley G.
Col. (M.C.), Station Hospital, Fort Hayes, Z18 OALR
Perry, Claude S.....Maj. (M.C.), 40 S. 3rd st., Z15 OP
Postle, C. D.....240 E. State, Z15 ALR
Prout, A. W.....380 E. Town, Z15 OALR
Sanor, Daniel G., Jr.....206 E. State, Z15 ALR
Silbernagel, C. E.....247 E. State, Z15 OALR
Stewart, James V. Capt. (M.C.), Columbus Army Air Base ALR
Stoutenborough, William A.....21 E. State, Z15 OALR
Thomas, Francis W.....327 E. State, Z15 OALR
Timberman, Andrew.....91 Hamilton Park, Z3 OALR
Troutman, Erwin W.....21 E. State, Z15 ALR

DAYTON

Adams, Eldridge S., Col. (M.C.),
Station Dispensary, Wright Field ALR
Beavis, James O.....900 Fidelity Medical bldg., Z2 ALR
Binkley, Roy S.....Fidelity bldg., Z2 OALR
Blackburn, W. J.....Reibold bldg., Z2 OALR
Boswell, Robert Echo.....Fidelity bldg., Z2 ALR
Clark, Willard C.....530 Fidelity bldg., Z2 OP
Driscoll, Frederick J.....Reibold bldg., Z2 ALR
Farmer, A. G.....Fidelity Medical bldg., Z2 OALR
Harris, H. B.....Fidelity Medical bldg., Z2 OALR
Kilbourne, P. H.....Fidelity Medical bldg., Z2 OALR
Thomas, Edward Robert.....60 Wyoming, Z9 OP

ELYRIA

Meredith, Lawrence Cornelius.....124 Middle av. ALR

FAIRFIELD

Craddock, Wm. H.....Lt. Col. (M.C.), Patterson Field ALR
Cunningham, John C.....Capt. (M.C.), Patterson Field OP
Steinberg, Martin R.....Maj. (M.C.), Patterson Field ALR

FINDLAY

Mowry, Ben.....First Nat'l Bank bldg. ALR

FREMONT

Riddell, Malcolm M.....112½ Front ALR

LAKEWOOD

Ellenberger, Carl.....14805 Detroit av., Z7 OP
Frackelton, Ralph James.....15701 Detroit av. ALR
McGannon, Francis Lambert.....14900 Detroit av. ALR
Merica, F. W.....14805 Detroit av., Z7 ALR
Miller, G. Leslie.....14805 Detroit av., Z7 OP

LIMA

Herr, Albert H.....Maj. (M.C.), 212 Steiner bldg. ALR
Stueber, F. G.....209 W. North OALR
Stueber, Paul J.....209 W. North OALR

LORAIN

Monosmith, O. B.....O. P. S. bldg. OALR

MANSFIELD

Garber, J. M.....14 N. Mulberry OALR
Wise, Ralph C.....News bldg. OALR

MARIETTA

Sauer, W. W.....231 Third OALR

OHIO — Continued

MARION
Brickley, Daniel W.....Uhler-Phillips bldg. OALR
Brickley, Daniel W., Jr.....Uhler-Phillips bldg. ALR

MIDDLETOWN
Williams, Walton H.....Castell bldg. OALR

MONROEVILLE
Bolman, Harold R.....6½ S. Main OP

NEWARK
Hatch, C. B.....3 W. Church OALR

SALEM
King, Lowell W.....470 E. State ALR

SIDNEY
Alvis, Raymond W.....Ohio bldg. OALR

SANDUSKY
Kramer, Julius C.....130 E. Market ALR

SHAKER HEIGHTS
Phillips, Josephine D.....16130 Parkland drive, Z20 OP

SPRINGFIELD
Burgman, James E.....8 W. Main ALR
Easton, J. C.....First Nat'l Bank bldg. OALR
Ernsting, Harry Carl.....8 W. Main st. OP
Hogue, D. W.....34 W. High OALR
Reuter, Carl H.....First Nat'l Bank bldg. OP
Yinger, Starling C.....First Nat'l Bank bldg. ALR

STEUBENVILLE
Quinn, John L.....401 Market ALR

TIFFIN
Porter, E. H.....85 Madison OALR

TOLEDO
Alderdyce, William W.....513 Madison av., Z4 OALR
Boice, Ralph E.....316 Michigan, Z2 OP
Cooper, James H.....316 Michigan st., Z2 OP
Denyes, Gerald Foster.....316 Michigan, Z2 ALR
King, Charles R.....Spitzer bldg., Z4 OALR
Lukens, Charles.....316 Michigan Z2 OALR
Lukens, John A.....316 Michigan, Z2 ALR
Minns, John Edward.....316 Michigan, Z2 ALR
Nippe, Leonard.....316 Michigan, Z2 OALR
Ravin, Louis C.....316 Michigan st., Z2 OP
Skow, John D.....2001 Collingwood av., Z2 OP
Steinfeld, A. L.....125 Fifteenth, Z2 OALR
Winger, Ira B.....709 Madison av., Z2 OALR

VAN WERT
Edwards, Gwilym Austin.....Home Guards bldg. ALR
Edwards, Stephen A.....Home Guards bldg. ALR

WARREN
Stimson, George W.....580 Perkins drive N. W. OALR

WILLOUGHBY
Waugh, J. M.....Ridge road, R. F. D. No. 1 OALR

YOUNGSTOWN
Goldcamp, Edward C.....Dollar Bank bldg., Z3 ALR
Goodwin, Vernon L.....509 Central Tower, Z3 ALR
Hall, Raymond A...Capt. (M.C.), 3521 Hopkins road ALR
Hartzell, Sol M.....260 Tod Lane OALR
Odom, Robert E.....Maj. (M.C.), 378 Lora av., Z4 ALR
Piercy, Frederick F.....Home Savings & Loan bldg., Z3 ALR
Walker, Orville J.....Home Savings & Loan bldg., Z3 ALR
Wenaas, Elmer J.....24 Wick av. OP

ZANESVILLE
Culbertson, L. R.....Masonic Temple OALR

OKLAHOMA

ADA
Peterson, William G.....106 E. 13th ALR

CHICKASHA
Morrissett, Leslie E., Maj. (M.C.), Borden Gen. Hospital ALR
Stroud, Sanders K., Capt. (M.C.), Borden Gen. Hospital OP

FORT SILL
McBean, Jas. B.....Maj. (M.C.), Station Hospital ALR
McLeod, John.....Maj. (M.C.), Cantonment Hospital OP

LAWTON
Schluer, E. P.....Maj. (M.C.), 710½ C st. ALR

MUSKOGEE
Coachman, Edwin H.....Manhattan bldg. ALR
Fullenwider, C. M.....Barnes bldg. OALR

NORMAN
Davis, Oscar, Capt. (M.C.), USN, U. S. Naval Hospital OALR
Freimuth, L. E., Lt. Comdr. (M.C.), U. S. Naval Hosp. ALR

OKLAHOMA CITY
Bonham, William L.....Medical Arts bldg., Z2 ALR
Cooper, F. Maxey.....Medical Arts bldg., Z2 OP
Coston, Tullos O.....Medical Arts bldg., Z2 OP
McHenry, L. Chester.....Medical Arts bldg., Z2 ALR
Macdonald, J. C.....301 N. W. 12th, Z3 ALR
Patterson, Robert U., Maj. Gen. U.S.A., (Retired)
700 N. W. 18th, Z3 ALR
Randel, Harvey O.....Medical Arts bldg., Z2 OP
Wails, Theodore G.....Medical Arts bldg., Z2 ALR
Watson, O. Alton.....1200 N. Walker, Z3 ALR

OKMULGEE
Dodge, W. M., Jr., Maj. (M.C.), Glennan Gen. Hospital OP
Tracewell, George Logan.....Commerce bldg. ALR

TULSA
Braswell, James C.....Medical Arts bldg., Z3 OALR
Cook, W. Albert.....Medical Arts bldg., Z3 OALR
Davis, Arthur Henry.....Medical Arts bldg., Z3 ALR
Haralson, Charles Herndon.....Medical Arts bldg., Z3 OP
Huber, Walter A.....Medical Arts bldg., Z3 OP
Mishler, D. L.....604 S. Cincinnati av., Z3 ALR
Roth, Albert W.....1616 S. Peoria, Z5 OALR
Smith, Ruric N.....Medical Arts bldg., Z3 ALR

WOODWARD
Eschenbrenner, John William.....A.A.F., Station Hosp. ALR

OREGON

ASTORIA
Spalding, Lansford M.....185 Fourteenth **ALR**

BAKER
Gordon, Glenn G.....Alexander bldg. **ALR**

EUGENE
Norton, Arthur H.....1162 Williamette **OP**

LA GRANDE
Bouvy, L. B.....Foley bldg. **OALR**
Ralston, Frank L.....Sacajawea Annex **OALR**

MEDFORD
Young, Clarence M.....Drawer 1506 **ALR**

PORTLAND
Bouvy, Harry M., 800 Pittock blk., 10th & Washington **OALR**
Boyden, Guy L.....1735 N. Wheeler, Z12 **ALR**
Browning, Andrew J.....Mayer bldg., Z5 **OP**
Carruth, Howard Ernest.....Medical Arts bldg., Z5 **OALR**
Davis, Ralph F.....Medical Arts bldg., Z5 **OALR**
Dykman, Augustus Bartram.....833 S. W. 11th, Z5 **OP**
Fenton, Ralph A.....Medical Arts bldg., Z5 **OALR**
Gaston, Ire E.....Medical-Dental bldg., Z5 **OALR**
Henton, George E.....812 S. W. Washington, Z5 **OP**
Jordon, Lewis W.....Medical Arts bldg., Z5 **ALR**
Karkeet, Robert B.....833 S. W. 11th av., Z5 **ALR**
Kiehle, Frederick A.....1020 Southwest Taylor, Z5 **OP**
Kistner, F. B.....Mayer bldg., Z5 **ALR**
Larsell, Olaf, Ph. D., Sc. D., University of Oregon
Lucas, Harold R.....Medical-Dental bldg., Z5 **OALR**
Lupton, Irving Martin.....Medical Arts bldg., Z5 **OALR**
Neely, Paul T.....Medical Arts bldg., Z5 **ALR**
Petheram, C. C.....Medical Arts bldg., Z5 **ALR**
Taylor, E. Merle.....Medical Arts bldg., Z5 **OP**
Weeks, John E. (Retired).....360 Edgecliff road, Z1 **OP**

SALEM
Clement, L. O.....Guardian bldg. **OALR**

WECOMA
Cutler, Franklin E..... **ALR**

PENNSYLVANIA

ABINGTON
Hughson, Walter.....Abington Memorial Hospital **A**

ALIQUIPPA
Thel, Henry C.....387 Franklin **ALR**

ALLENTOWN
Hertz, William J.....125 N. 8th **ALR**
Phillips, Samuel A.....137 N. 8th **OP**
Rothenberger, Marvin K.....206 N. 8th st. **ALR**
Uhler, S. Mann.....104 N. 8th **ALR**

ALTOONA
Buzzard, Josiah F.....1110 Thirteenth av. **OALR**
Glover, Lewis P.....1200 Fourteenth av. **OP**
Hogue, John Dan.....909 Lexington av. **ALR**

ARDMORE
Bostwick, D. S.....106 Linwood av. **ALR**
Furlong, Thomas F., Jr.....Times Medical bldg. **ALR**
Longaker, Edwin P.....41 E. Montgomery av. **ALR**

BEAVER
Crumrine, Norman Ross.....595 Third **OALR**

BEAVER FALLS
Baldwin, Thomas M.....222 Brodhead bldg. **ALR**
Reed, Elmer McCready, Capt. (M.C.), c/o 2835-4th av. **ALR**

BETHLEHEM
Chase, W. D.....230 E. Broad **ALR**

BRYN MAWR
McAndrews, Leo F.....Mermont-Plaza **OP**
Snyder, George Gordon.....Mermont Apts **ALR**

BUTLER
Beck, F. W...Capt. (M.C.), Deshon General Hospital **ALR**
Goehring, Donald Everett.....Butler S. & T. bldg. **ALR**
Hunt, Guy Anderson.....Butler Co. Nat'l Bank bldg. **OP**
May, Frederick T., Maj. (M.C.), Deshon General Hospital **OP**
Mobley, Marion R., Lt. Col.. (M.C.), Deshon Gen. Hosp. **ALR**

CARBONDALE
Marmelstein, Maurice.....1st Nat'l Bank bldg. **OP**

CARLISLE
George, Forney P.....118 S. Hanover st. **ALR**
Moser, George P.....Capt. (M.C.), 76 Willow st. **ALR**
Stoner, Donald D.....156 W. Pomfret st. **ALR**

CHAMBERSBURG
Himelfarb, H. M.....Capt. (M.C.), R. F. D. No. 4 **ALR**

CHARLEROI
Rowland, Ivo E.....5th and McKean **ALR**

CHESTER
Cross, G. H.....525 Welsh **OP**

CLEARFIELD
Shaffer, Harry Gard.....225 Cherry **ALR**

CONNELLSVILLE
Harrison, Fred H.....Second Nat'l Bank bldg. **OALR**
McLellan, Thomas G.....First Nat'l Bank bldg. **OP**

DANVILLE
Davison, Francis Wells.....Geisinger Hospital **ALR**
Jacobs, Clyde Hillock.....Geisinger Memorial Hosp. **OP**
Nebinger, Reid.....16 W. Market **ALR**

DREXEL HILL
Weber, H. C.....Capt. (M.C.), U.S.N., 112 Mason av. **OALR**

DU BOIS
Beals, C. Wearne.....28 W. Scribner av. **ALR**
Tyson, John McClure.....3 S. Brady **ALR**

EASTON
Bloom, C. Hugh.....65 N. Third **ALR**

ERIE
Delaney, James H.....138 W. 9th **OP**
Donovan, John E.....265 W. 10th st. **ALR**
Fust, John H.....231 W. 7th **OALR**
Jackson, J. D.....232 W. 8th **OALR**
Russell, J. A. Merle.....813 Sassafras **OALR**
Schlindwein, G. William.....138 W. 9th **OALR**
Shreve, Owen M.....162 W. 8th **OP**
Simmons, J. Thomas.....316 W. 10th st. **OP**

PENNSYLVANIA — Continued

FRANKLIN
Jobson, G. B.....West Park and Liberty OALR
Nordstrom, Chester Albert.....Exchange Bank bldg. ALR

GREENSBURG
Fairing, John W.....122 N. Main OALR
McKee, Claud W.....Coulter bldg. OALR

HARRISBURG
Dailey, Gilbert L.....618 N. 3rd OALR
Gracey, George F.....209 State OALR
Zimmerman, J. Landis.....207 State OP

HAZLETON
Reiche, O. C.....Markle Bank bldg. OP

HUNTINGDON
Hunt, William T., Jr.....514 Penn OALR

INDIANTOWN GAP MILITARY RESERVATION
Krieger, Alexander A.....Maj. (M.C.), Station Hosp. OP

JENKINTOWN
Follweiler, Frank L.....423 York rd. ALR
Kauffman, M. Luther.....Medical Arts bldg. OP

JOHNSTOWN
Griffith, Harold M.....Johnstown Bank & Trust bldg. OP
Harris, C. M.....Johnstown Bank & Trust bldg. OALR
Hays, Charles E.....U. S. Bank bldg. ALR
McCloskey, Paul.....338 Locust ALR
Parker, Ray.....U. S. Bank bldg. ALR

LANCASTER
Fulton, Harry C.....339 N. Duke OP
LeFevre, Willis H.....234 N. Duke ALR
Smith, Ernest Gerard.....339 N. Duke OP

LATROBE
Mather, Homer Raymond.....224 Depot st. ALR

LEBANON
Strickler, Alfred D.....26 N. 9th st. OP

LEWISTOWN
Allis, Paul M.....6 N. Brown ALR

MEADVILLE
Hazen, S. Frank.....231 Chestnut ALR

MEDIA
Lewis, Fielding O.....(Retired) ALR

MONESSEN
Gemmill, Walter D.....130 Seventh ALR

MORRISVILLE
Moriconi, Albert F.....Maj. (M.C.), 104 E. Ferry rd. ALR

NEW KENSINGTON
Steim, J. M.....First Nat'l Bank bldg. OP

NORRISTOWN
Beideman, Joseph Ellsworth..Montgomery Trust Arcade OP
LaClair, Chas. H.....Lt. Col. (M.C.), 820 W. Marshall ALR

OIL CITY
Magee, F. Earle.....222 Seneca ALR

PHILADELPHIA
Adler, Francis Heed.....313 S. 17th, Z3 OP
Annon, W. T.....235 S. 15th, Z2 ALR
Appleman, L. F.....308 S. 16th, Z2 OP
Babbitt, James A.....1912 Spruce, Z3 ALR
Baldwin, Gertrude W.....255 S. 17th st., Z3 OP
Balentine, P. L.....1524 Chestnut, Z2 OP
Ball, Simon..Lt. Comdr. (M.C.), U. S. Naval Hosp., Z45 ALR
Barlow, Aaron.....1307 Spruce, Z7 OP
Batson, Oscar V.....133 S. 36 st., Z4 ALR
Beam, A. Duane....Lt. (M.C.), U. S. Naval Hosp, Z45 OP
Berner, George Emil.....5430 Greene, Z44 OP
Bertolet, John Allen.....1530 Locust, Z2 ALR
Blair, Mortimer W.....N. E. cor. 20th and Chestnut, Z3 OP
Bricker, Sacks.....1101 W. Wyoming av., Z40 ALR
Bridgett, Frank Aloysius.....332 Chestnut st., Z4 ALR
Briglia, Frank J.....Lt. (M.C.), 819 Wharton, Z47 ALR
Brumm, Seth A.....Medical Arts bldg., Z3 OALR
Burns, L. J.....1930 Chestnut, Z3 ALR
Butt, Miriam M.....2045 Chestnut, Z3 OP
Campbell, Sherburne
Lt. Comdr. (M.C.), U. S. Naval Hospital, Z45 OP
Carpenter, Chapin.....1930 Chestnut, Z3 OP
Cierf, Louis Henry.....1530 Locust, Z2 ALR
Coates, George M.....1721 Pine, Z3 ALR
Cohen, Herman B.....255 S. 17th, Z3 ALR
Cohen, Louis B.....1320 Arrott, Z24 OP
Cohen, Samuel.....1920 Pine, Z3 ALR-PI S
Cowan, Alfred.....1930 Chestnut, Z3 OP
Creighton, William J.....37 S. 20th, Z3 OP
Davis, T. Carroll.....3128 N. Broad, Z32 ALR
Davis, Warren B.....135 S. 18th, Z3 ALR-PI S
Deichler, L. Walter.....1930 Chestnut, Z3 OALR
Dickson, Robert L.....4803 Penn, Z24 ALR
Dintenfass, Henry.....1305 Spruce, Z7 ALR
Doiowitz, David A., Lt. (M.C.).....5129 No. 12 st. ALR
Donnelly, Edward J.....1321 Spruce, Z7 OP
Dublin, George Joseph.....1930 Chestnut, Z3 OP
Elfman, Louis Kenneth.....6330 Lebanon av., Z31 ALR
Ersner, Matthew S.....1915 Spruce, Z3 ALR
Fewell, A. G.....1924 Pine, Z3 OP
Fox, C. Calvin.....1806 Spruce, Z3 ALR
Fry, W. E.....1819 Chestnut, Z3 OP
Gibson, Glen G.....255 S. 17th, Z3 OP
Gordon, William.....1721 Pine, Z3 ALR
Gowens, Henry L. Jr.....2217 Delancey st., Z3 OP
Hawman, Erle G.....4051 N. Broad, Z40 OP
Hebbie, Howard M.
Lt. (M.C.), U. S. Naval Hospital, Z45 ALR
Heed, C. R.....1205 Spruce, Z7 OP
Hewson, William.....2031 Locust st. ALR
Hirst, Otto C.....255 S. 17th, Z3 ALR
Hitschler, William Jacob....8840 Germantown av., Z18 ALR
Hoffman, N. Fulmer, Jr.....2035 Delancey Pl., Z3 ALR
Houser, Karl M.....2035 Delancey st., Z3 ALR
Husik, D. N.....1820 Spruce, Z3 ALR
Ivy, Robert H.....1930 Chetnut, Z3 ALR-PI S
Jackson, Chevalier.....255 S. 17th, Z3 L
Jackson, Chevalier L.....255 S. 17th, Z3 L
Kasper, Kelvin A.....135 S. 18th, Z3 ALR
Katz, Harry Kolman.....1206 Cottman st., Z11 OP
Kaufman, A. Spencer.....1923 Spruce, Z3 ALR
Kelly, George F. J.....37 S. 20th, Z3 OP
Krauss, Frederick.....1930 Chestnut, Z3 OALR
Krewson, William Egbert, 3rd.....1930 Chestnut, Z3 OP
Lawrence, Granville A.....Medical Arts bldg., Z3 OP

PENNSYLVANIA — Continued

Lehrfeld, Louis.....1321 Spruce, Z7 OP
Leopold, Irving H.
University Hospital, University of Pennsylvania, Z4 OP
Lillie, W. Ivan.....255 S. 17th, Z3 OP
Loftus, John Edward.....1930 Chestnut, Z3 ALR
Lukens, Robert McD
Suite 1001, Medical Arts bldg., 16th and Walnut, Z3 ALR
Luongo, R. A.....2054 Locust, Z3 ALR
McCarthy, C. T.....1810 Pine, Z3 ALR
McDonald, P. Robb.....Maj. (M.C.), 255 S. 17th, Z3 OP
Mackenzie, George W.....269 S. 19th, Z3 OALR
Mann, Benjamin H.....1930 Chestnut, Z3 OP
Miller, M. Valentine, 114 W Phil-Ellena (Mt. Airy), Z19 ALR
Mitchell, E. K.....710 W. Lehigh av., Z33 ALR
Mullen, Carroll R.....2025 Locust, Z3 OP
Olsho, S. L.....235 S. 15th, Z2 OALR
Persky, Abram H.....1737 Chestnut, Z3 ALR
Pryor, Charles Allen.....1910 Spruce, Z3 ALR
Reese, Warren S.....1901 Walnut, Z3 OP
Richardson, George A.....1532 Overington, Z24 ALR
Ridpath, Robert F.....1737 Chestnut, Z3 LR
Rommel, John C.....4601 N. Broad, Z40 OP
Rudolphy, Jay Besson.....230 S. 21st, Z3 OP
Sartain, Paul J.....2006 Walnut, Z3 OP
Scarlett, Hunter W.....230 S. 21st, Z3 OP
Seltzer, Albert Pincus.....1332 N. Franklin st., Z22 ALR
Shannon, C. E. G.....1930 Chestnut, Z3 OP
Shipman, James S.....255 S. 17th, Z3 OP
Shuster, Benjamin Harrison.....1824 Pine, Z3 ALR
Simkins, James J.....1737 Chestnut, Z3 ALR
Skillern, S. R., Jr.....1734 Pine, Z3 ALR
Sloane, Henry O.....1717 Pine, Z3 OP
Spaeth, Edmund B.....1930 Chestnut, Z3 OP-PI S
Spencer, W. H.....1830 S. Rittenhouse sq., Z3 ALR
Stiles, Charles M.....334 W. Wiona av., Z44 OP
Stockwell, Eunice LeBaron.....2018 Pine st., Z3 OP
Sutherland, William S.....1930 Chestnut, Z3 OP
Swalm, Charles Joseph.....1330 W. Rockland, Z41 ALR
Tassman, Isaac S.....Medical Arts bldg., Z3 OP
Towson, Charles E.....Germantown Prof. bldg., Z44 ALR
Tucker, Gabriel.....250 S. 18th, Z3 LB
Wagers, Arthur J.....1429 Spruce, Z2 ALR
Waldman, Joseph
Maj. (M.C.), Park Manor Apts., 4000 Girard av., Z4 OP
Walsh, Thomas J.....2059 Parrish, Z30 ALR
Washick, Frank.....501 Cottman st., Z11 ALR
Weinstein, M. A.....1530 Locust, Z2 ALR
Whelan, G. L.....2100 Walnut, Z3 ALR
Whelan, William F.....2004 Walnut, Z3 ALR
Williams, Horace J.....5908 Greene (Germantown), Z44 ALR
Winston, Julius.....2021 Spruce, Z3 ALR
Wolf, Lewis R.....3474 Frankford av., Z34 OP
Zacks, Myron Albert.....1737 Chestnut, Z3 ALR
Zentmayer, William.....1930 Chestnut, Z3 OP

PHOENIXVILLE

Abbott, A. J.
Capt. (M.C.), Valley Forge General Hospital OP
Boyd, Charles Wilson, Maj. (M.C.),
Valley Forge Gen. Hospital OP
Greear, James N., Jr., Lt. Col. (M.C.),
Valley Forge General Hospital OP
Mountain, Edward R., Capt. (M.C.),
Valley Forge General Hospital ALR

PITTSBURGH

Bernatz, C. F.....Park bldg., Z22 OP
Billings, Robert J.....500 Penn av., Z22 OP
Blair, W. W.....Woodland rd., Z6 OP
Boaz, Emmett D.....3710 Fifth av., Z13 ALR
Carson, W. E.....Jenkins Arcade, Z22 OP
Conway, John M.....Jenkins Arcade, Z22 ALR
Cooper, Roy C.....510 S. Aiken av., Z6 ALR
Curry, Glendon E.....Diamond Bank bldg., Z22 OP
Day, Kenneth M.....121 University pl., Z13 ALR
De Stio, Daniel Stephen.....Highland bldg., Z6 ALR
Dunbar, John Charles.....Empire bldg., Z22 OP
Fischer, N. Arthur.....Union Trust bldg., Z19 ALR
Friedman, Louis Leonard.....6078 Jenkins Arcade, Z22 ALR
Gross, Julius E.....3401 Fifth av., Z13 ALR
Hagemann, John A.....Highland bldg., Z6 ALR
Hamilton, Richard G.....Medical Arts bldg., Z13 ALR
Hampsey, Joseph William.....806 May bldg., Z22 ALR
Jennings, Charles W.....121 University pl., Z13 OP
Krebs, Adolph.....500 Penn av., Z22 OP
Larimore, Fred Campbell.....500 Penn av., Z22 OP
Linn, J. G.....Jenkins Arcade, Z22 OP
McCaslin, Murray F.....Union Trust bldg., Z19 OP
McCollough, T. B.....121 University pl., Z13 ALR
McCready, J. Homer.....121 University pl., Z13 ALR
MacLachlan, A. A.....May bldg., Z22 ALR
Markel, J. Clyde.....806 Pitt Bank bldg., Z22 OP
Marshall, Watson.....121 University pl., Z13 ALR
Miller, A. Boyd, 1006 Prof. bldg., 429 Penn av., Z22 ALR
Moon, Seymour B.....429 Pennsylvania av., Z22 OP
Morgan, Irving Joseph.....Jenkins Arcade bldg., Z22 OP
Pachtman, Isadore.....Jenkins Arcade bldg., Z22 OP
Patterson, Ellen J.....(Retired), 504 Neville, Z13 ALR
Perrone, Joseph Addison.....Mercy Hospital, Z19 ALR
Plumer, John S.....121 University pl., Z13 OP
Ratner, Simon Harry.....3401 Fifth av., Z13 ALR
Reed, Charles L.....Jenkins Arcade, Z22 OP
Rhodes, David Henry.....Clark bldg., Z22 OP
Rimer, Frank H.....Jenkins Arcade, Z22 ALR
Rohm, Robert Franklin.....7026 Jenkins Arcade Z22 OP
Shaffer, D. Howard.....7036 Jenkins Arcade, Z22 OP
Shepard, Warren Brundage.....Jenkins Arcade, Z22 ALR
Shuman, G. H.....351 Fifth av., Z22 OP
Simpson, John Reid.....Medical Arts bldg., Z13 ALR
Statti, Louis William.....Jenkins Arcade, Z22 OP
Stieren, Edward.....Union Trust bldg., Z19 OP
Taylor, M. C.....May bldg., Z22 OP
Thorpe, H. E.....Jenkins Arcade, Z22 OP
Todd, Grover Cleveland.....Jenkins Arcade, Z22 ALR
Turner, H. H.....Clark bldg., Z22 OP
Weisser, E. A.....May bldg., Z22 OP

PITTSTON

Gagion, Thomas Reed.....23 Broad OP
Gallizzi, Vincent D.....51 S. Main st. ALR

POTTSTOWN

Hancox, Cecil William.....Bahr Arcade bldg. ALR

POTTSVILLE

Cress, William J.....318 W. Market ALR
Mengel, Sterling Frederic
Lt. Comdr. (M.C.), 612 W. Market OP

READING

Bankes, Claude W.....212 N. 6th OP
Craig, Paul C.....232 N. 5th OP

PENNSYLVANIA — Continued

High, Isaac B.....326 N. 5th ALR
Landis, James E.....232 N. 6th ALR
Penta, Michael J.....312 N. 5th ALR
Rhode, Homer J.....220 N. 6th OP
Rhode, Solon L.....238 N. 6th OALR
Strause, Harold L.....826 N. 5th st. ALR
Wiest, Philip R., Capt. (M.C.),.....1036 Franklin st. ALR
Wotring, John M.....220 N. 6th OP

ROCHESTER
Miksch, Henry Fred.....209 S. Park ALR

SCRANTON
Bishop, Frederick J.....Medical Arts bldg., Z3 ALR
Corser, J. B.....415 Mulberry, Z3 OP
Curtin, Eugene A.....506 Spruce, Z3 OALR
Jordan, James Shannon.....Medical Arts bldg., Z3 OP
Williams, M. M.....Medical Arts bldg., Z3 OP

SHARON HILL
Tomlinson, John Wesley
Lt. Comdr. (M.C.), 900 Chester Pike OP

SHENANDOAH
Monohan, James J.....31 S. Jordin ALR

SUMMIT HILL
Bonner, William Richard.....3 W. White st. OP
Houser, Ben P.....Capt. (M.C.), 18 W. Holland st. ALR

TURTLE CREEK
Hunter, James F.....503 Penn av. ALR

UPPER DARBY
Kennedy, Patrick James.....6910 Market OP
Moore, George H.....101 Long Lane OALR
Rankin, Charles A.....Ludlow and Heather road OP

WARREN
Anderson, Edwin R.....220 Liberty OALR
Ball, Michael V.....316 Hazel OALR

WASHINGTON
McCullough, Clarence Joseph....Washington Trust bldg. ALR
McKennan, James W.....Washington Trust bldg. OP
McMurray, J. B.....Washington Trust bldg. OALR

WILKES-BARRE
Buckman, Lewis T.....83 S. Franklin OALR
Buckman, Samuel T.....70 S. Franklin OALR
Connole, Joseph V.....Miners Nat'l Bank bldg. OP
Kistler, O. F.....43 N. Franklin OP AR
Marsden, W. C.....99 N. Franklin OALR
Schappert, N. Louis.....59 S. Washington OALR

WILLIAMSPORT
Decker, Philip Harold.....25 W. 3rd st., Z5 ALR

YORK
Fisher, Lawrence Christian.....469 W. Market OP
Grove, Bruce Alexander.....141 E. Market st. OP
Klinedinst, John Fred.....238 S. George st. OPA
Podboy, August J.....211 S. George st. ALR

RHODE ISLAND

DAVISVILLE
Weiss, J. Allen, Lt. Comdr. (M.C.)
U. S. Naval Construction Training Center, B. O. Q.-04 ALR

E. GREENWICH
Dinan, Thomas Edward
Lt. Comdr. (M.C.), 44 Beechwood Drive, Bayridge ALR

NEWPORT
Jacoby, Douglas P. A.....106 Touro OAL
Meek, R. E.....Comdr. (M.C.), U. S. Naval Hospital OP
Moyle, Eugene H.
Lt. Comdr. (M.C.), U. S. Naval Hospital ALR

PROVIDENCE
Adams, Frank M.....122 Waterman, Z6 ALR
Bolotow, Nathan A.....126 Waterman, Z6 ALR
Dimmitt, Frank W.....78 Waterman, Z6 OALR
Grossman, Herman P.....210 Angell, Z6 OP
Harvey, N. Darrell.....112 Waterman, Z6 OALR
McCurdy, Gordon Judson.....198 Angell, Z6 ALR
MacCardell, Frank Cameron.....193 Waterman, Z6 ALR
Messinger, H. C.....210 Angell, Z6 OP
Muncy, William M.....162 Angell, Z6 OALR
Porter, L. B.....199 Thayer, Z6 OALR
Winkler, Herman A.....224 Thayer, Z6 ALR

QUONSET POINT
Cammisa, James J. V., Comdr. (M.C.),
U. S. Naval Air Station, Med. Dept. OALR

SOUTH CAROLINA

CAMP CROFT
Fairfax, Kenneth T.....Maj. (M.C.), Station Hospital OP
Holmes, Will W., Col. (M.C.),.....Station Hospital ALR

CHARLESTON
Asbill, David S., Capt. (M.C.),....C.P.E. Station Hosp. OP
Evatt, Clay W.....112 Rutledge av., Z16 ALR
Hanckel, Richard White, Jr.....96-A Bull st., Z16 ALR
Hope, Robert Meek.....108 Rutledge av., Z16 ALR
McMurray, John S., Capt. (M.C.),
Station Hospital, C.A.A.B. ALR
Pritikin, Roland I.
Lt. Col. (M.C.), Stark General Hospital OP
Salmon, D. L., Maj. (M.C.),....Stark General Hospital ALR
Sitney, Julian J., Lt. (M.C.), Station Hospital, C.A.A.B. OP
Smith, Josiah Edward.....60 Meeting, Z1 OALR
Townsend, John Ferrars.....85 Wentworth, Z6 OALR
Trueman, Robert Heiserman
Maj. (M.C.), Stark General Hospital OP
Wood, Edward C., Maj. (M.C.),
Station Hospital, Charleston, A.A.B. OP

COLUMBIA
Bristow, Walter J.....1517 Hampton, Z29 OALR

FORT JACKSON
Almour, Ralph, Maj. (M.C.),..74th General Hospital ALR
Herzog, Max, Capt. (M.C.).....Station Hospital OP
Schlessel, Louis, Maj. (M.C.), 95th General Hospital ALR
Smith, Jay D.
Maj. (M.C.), Box 6, Station Hospital No. 2 ALR
Tinkess Donald E., Maj. (M.C.), Station Hosp., Box 42 OP

SOUTH CAROLINA — Continued

GREENVILLE
Sanders, John Levis.....222 N. Main ALR

PARRIS ISLAND
Feldman, Irvin, Lt. Comdr. (M.C.), USNR
Post Medical Detachment, Marine Barracks ALR
Hutchison, Lloyd S.
Lt. Comdr. (M.C.), U. S. Naval Hospital ALR

ROCK HILL
Macdonald, Roderick.....330 E. Main ALR

SUMTER
Walker, R. M., Lt. (M.C.), USNR, 201 N. Main st. ALR

SOUTH DAKOTA

ABERDEEN
Bunker, Paul Gordon.....Newberry bldg. ALR
Rudolph, Edward.....Newberry bldg. OP

BROOKINGS
Kershner, C. M.....Watson Clinic OP

MITCHELL
Mabee, Oscar Judson.....Medical Arts bldg. ALR

PIERRE
Robbins, Clarence Edward.....111 S. Huron av. ALR

SIOUX FALLS
Butler, Harry, Lt. Col. (M.C.),
Sioux Falls Army Air Field ALR
Geeseman, George Robert, Capt. (M.C.),
Station Hospital, AAFTTC OP
Gregg, John B.....Security Nat'l Bank bldg. OALR
Keller, S. A.....303 S. Minnesota av. OALR

YANKTON
Hohf, Julius A.....Clinic bldg. OALR

TENNESSEE

CAMP FORREST
Hinderer, Kenneth H.....Capt. (M.C.), Station Hosp. ALR
Kincannon, LeRoy T., Maj. (M.C.), 216th General Hosp. OALR

CHATTANOOGA
Hogshead, J. M.....602 Georgia av., Z3 OALR
Isbell, D.....552 McCallie av., Z3 ALR
Lassiter, Chester L.....Hamilton Nat'l Bank bldg., Z2 ALR
Lawwill, Stewart.....Medical Arts bldg., Z3 OALR
Long, Samuel Herman.....Medical Arts bldg., Z3 ALR
Steele, Willard.....721 Broad, Z2 OALR

CLARKSVILLE
Wilson, Paul Edwin.....408 Franklin OP

CLEVELAND
Ferguson, E. R.....404 Inman st. ALR

CROSSVILLE
Mercer, Ray.....Lt. Col. (M.C.), P.W.I.C. Hospital OALR

DYERSBURG
Brewer, John D.....Baird Brewer General Hospital OALR

JOHNSON CITY
Jones, U. G.....Cor. Market and Boone ALR
McKee, Thomas Preston.....Jones Hospital ALR

KINGSPORT
Hodge, James Vernon.....105 Market ALR

KNOXVILLE
Cunningham, Henry Kelso.....Medical Arts bldg., Z01 ALR
Kincaid, J. H.....421 W. Church av., Z42 OALR
Leach, Robert S.....Medical Arts bldg., Z2 OP
Le Tellier Forrest S.....Doctors bldg., Z30 OP
Miller, Edwin Ephron.....Medical Arts bldg., Z01 ALR
Potter, William W.....601 Walnut, Z30 OALR

MARYVILLE
Burchfield, George W.....309 Court ALR

MEMPHIS
Anderson, R. G.
Maj. (M.C.), Kennedy General Hospital, Z15 OP
Anthony, Daniel Harbert.....Exchange bldg., Z3 OP
Blassingame, C. B.....899 Madison av., Z3 ALR
Blue, J. B.....62 N. Main, Z3 OALR
Ellett, E. C.....Exchange bldg., Z3 OP
Evans, S. S.....Physicians & Surgeons bldg., Z3 ALR
Farrington, P. M.....89 Porter bldg., Z3 ALR
Harris, J. Harley..Methodist Hospital Doctors bldg., Z4 ALR
Levy, Louis.....Commerce Title bldg., Z3 ALR
Lewis, A. C.....130 Madison av., Z3 OALR
Lewis, Philip Meriwether.....Exchange bldg., Z3 OP
McKinney, James Wesley.....Echange bldg., Z3 OP
Minor, H. F.....Commerce Title bldg., Z3 OALR
Qualls, H. W.....1635 Exchange bldg., Z3 ALR
Rychener, Ralph O.....130 Madison av., Z3 OP
Sanders, Sam H.....1089 Madison av., Z3 ALR
Seligstein, Milton B.....Union Planters Bank bldg., Z3 OALR
Shea, J. J.....1018 Madison av., Z3 ALR
Simpson, W. Likely....Physicians & Surgeons bldg., Z3 OALR
Stanford, J. B.....899 Madison av., Z3 OP
Stinson, William David.....899 Madison av., Z3 ALR

NASHVILLE
Bryan, James Leslie.....Bennie-Dillon bldg., Z3 OALR
Cullom, M. M.....Bennie-Dillon bldg., Z3 OALR
Hollabaugh, Charles F.....Shackleford road, Z4 OP
Irwin, Thomas M., Maj. (M.C.), Thayer General Hospital ALR
Kennon, W. G.....Doctors bldg., Z3 OALR
Maness, Guy Milford.....119 7th av. N., Z3 ALR
Orr, Eugene.....Doctors bldg., Z3 ALR
Sharp, Benjamin S., Maj. (M.C.), Thayer Gen. Hosp., Z5 OALR
Simpson, G. Victor, Lt. Col. (M.C.),
Nashville Army Air Center, Z4 OP
Sullivan, Robert E.....Doctors bldg., Z3 OP
Warner, Robert Jay.....706 Church, Z3 OP
Wilkerson, William W., Jr.....Bennie-Dillon bldg., Z3 ALR

OAK RIDGE
Bryant, Frank L., Maj. (M.C.), 0237676, P. O. Box 388 ALR

SMYRNA
Aronson, Sidney S., Maj. (M.C.), Station Hosp. S.A.B. ALR

TEXAS

AMARILLO

Crume, John James.....724 Polk **ALR**
Duncan, Robert Austin.....Amarillo bldg. **ALR**
McGillicuddy, O. B.....Maj. (M.C.), Station Hospital **ALR**
Merz, Earl H., Capt. (M.C.), 0491970,
Amarillo Army Air Field **OP**
Streit, August J.....724 Polk **ALR**

AUSTIN

Key, Samuel N.....Norwood bldg., Z16 **OALR**
Lippmann, Otto.....406 Scarbrough bldg., Z22 **OP**
Robison, J. T.....Norwood bldg., Z16 **ALR**

BIG SPRING

Hedemark, Norman G., Maj. (M.C.),
Station Hospital, A.A.F.B.S. **ALR**
Malone, P. W.....811 Main **ALR**

BROWNWOOD

Brown, Wilbur M.....Lt. Col. (M.C.), 1604 Vincent **OP**

CAMP BOWIE

Lauppe, Frederick A., Capt. (M.C.)..29th Field Hosp. **OP**

CAMP HOWZE

Heare, Louis C.....Lt. Col. (M.C.), Station Hospital **OP**
Smith, Herschel S., Maj. (M.C.),
Med. Det. 411th Inf., A. P. O. 470 **OP**

CAMP SWIFT

Green, Maurice L.....Maj. (M.C.), Station Hospital **OP**
Ho, Wayne Y. H., Capt. (M.C.), 0-534488, Sta. Hosp. **ALR**
King, George L., Jr., Lt. Col. (M.C.), 111th Evac. Hosp. **OP**

CORPUS CHRISTI

McNair, Sterling S., Lt. Comdr. (M.C.),
234 E. Vanderbilt **OP**

DALLAS

Barr, William T.....1327 Medical Arts bldg., Z1 **ALR**
Brannin, Daniel.....Medical Arts bldg., Z1 **OP**
Cary, E. H.....Medical Arts bldg., Z1 **OALR**
Cox, Kelly.....Medical Arts bldg., Z1 **OP**
Daniel, Ruby K.....Medical Arts bldg., Z1 **OP**
Harrington, Silas Frederick.....Medical Arts bldg., Z1 **ALR**
Knowles, W. Mood.....Medical Arts bldg., Z1 **ALR**
McCullough, Malcolm Kelley....Medical Arts bldg., Z1 **OP**
McLaurin, J. G.....4710 Munger av., Z4 **OALR**
Marchman, Oscar M.....5328 Live Oak, Z6 **ALR**
Nelson, Leo A.....4105 Live Oak, Z4 **ALR**
Newton, F. H.....Medical Arts bldg., Z1 **OALR**
Quinn, Lester Hale.....4105 Live Oak, Z4 **OP**
Scarney, Herman D., Comdr. (M.C.), USNR,
Naval Air Station **OP**
Sellers, L. M.....1719 Pacific av., Z1 **ALR**
Singleton, John Dudley.....1719 Pacific av., Z1 **ALR**
Wilkinson, Wallace Bond.....521 E. 10th, Z8 **ALR**

DEL RIO

Mertins, Paul S., Jr.....Capt. (M.C.), A.A.F.A.F.S. **ALR**

ELLINGTON FIELD

Cone, Alfred J., Maj. (M.C.), Station Hospital A.A.F.T.S. **ALR**

EL PASO

Clark, James W.....Maj. (M.C.), 3247 Louisville **OP**
Filmer, George A.
Maj. (M.C.), Wm. Beaumont General Hospital **OP**
Hall, S. S., Lt. Col. (M.C.),
51st General Hospital, Fort Bliss **ALR**
Schuster, Frank P.....First Nat'l Bank bldg. **OALR**
Schuster, S. A.....First Nat'l Bank bldg. **ALR**
Spearman, Maurice P.....First Nat'l Bank bldg. **ALR**
Vandevere, William E.....First Nat'l Bank bldg. **OALR**

FORT SAM HOUSTON

McKay, Edward D., Maj. (M.C.), Brooke Gen. Hosp. **OP**

FORT WORTH

Goodman, Thomas L.....1601 Clover Lane, Z7 **ALR**
Gough, Roy H.....600 W. 10th, Z2 **OALR**
Jackson, Atras E.....Medical Arts bldg., Z2 **OP**
Richardson, James Joseph,
Cook Hospital and Beall Clinic, Z3 **ALR**
Schenck, C. P.....Medical Arts bldg., Z2 **OALR**

GAINSVILLE

Mead, Ernest Charles.....106½ E. California **ALR**

GALVESTON

McReynolds, Geo. S., Jr.....927 Strand **ALR**
Potter, William Bently
University of Texas Medical College **OP**
Sykes, Clarence Sherbourne.....2201 Market **OP**

GREENVILLE

Gill, E. K., Lt. Col. (M.C.), Station Hospital
Majors Army Air Field **ALR**
Votaw, Robert E.....Maj. (M.C.), Station Hospital **ALR**
Ward, James W.....2607 Lee **ALR**

HONDO

Hitz, John B.....Maj. (M.C.), A.A.F.N.S. **OP**

HOUSTON

Alexander, Charles S.....Med. Arts bldg., Z2 **OP**
Barrett, John H.....1904 Walker av., Z2 **ALR**
Bressler, J. L.....Medical Arts bldg., Z2 **OP**
Cody, Claude C.....1304 Walker av., Z2 **ALR**
Daily, Louis.....Medical Arts bldg., Z2 **OALR**
Daily, Ray K.....Medical Arts bldg., Z2 **OALR**
Dickson, J. Charles.....Medical Arts bldg., Z2 **ALR**
Elles, Norma B.....Niels Esperson bldg., Z2 **OALR**
Foster, J. H.....1304 Walker av., Z2 **ALR**
Goar, Everett Logan.....1300 Walker av., Z2 **OP**
Griffey, Edward W.....Medical Arts bldg., Z2 **OALR**
Haden, Henry C.....1914 Travis, Z3 **OP**
Harris, Herbert Houston
Maj. (M.C.), Mellie Esperson bldg., Z2 **ALR**
Janse, Hector Mason.....3717 Main, Z4 **ALR**
Klotz, William F., Lt. Col. (M.C.),
5701 Jackson, Apt. 1007 **ALR**
Logue, Lyle J.....1304 Walker av., Z2 **ALR**
Ralston, Wm. W.....1304 Walker av., Z2 **OP**
Robison, J. M.....1304 Walker av., Z2 **ALR**
Spencer, Walter C. E.....Medical Arts bldg., Z2 **OP**
Stough, John Trew.....Medical Arts bldg., Z2 **OP**

KELLY FIELD

Moran, Charles T.....Maj. (M.C.), Station Hospital **OP**

TEXAS — Continued

LONGVIEW
Hurst, V. Reeves.....315 N. Center st. OALR
Jaffe, Benjamin
Capt. (M.C.), Harmon General Hospital. OP

LUBBOCK
Malone, F. B.....2609 19th ALR

McKINNEY
Lowenthal, Gerson
Maj. (M.C.), Ashburn General Hospital ALR
Whitsell, Fay M., Capt. (M.C.), Ashburn Gen. Hospital OP

MARFA
Gump, Millard E., Maj. (M.C.),
A.A.F.A.F.S.—M.A.A.F. Station Hosp. OP

MYRA
Maxwell, Hal W.....Lt. (M.C.), USNR OP

PAMPA
High, Clifton E.....Combs Worley bldg. OP

PARIS
Hunt, Thomas Ewell.....First Nat'l Bank bldg. ALR

PORT ARTHUR
Vaughan, Benjamin H.....Adams bldg. ALR

RANDOLPH FIELD
Byrnes, Victor A., Col. (M.C.),
School of Aviation Medicine OP
Campbell, Paul A.
Lt. Col. (M.C.), School of Aviation Medicine ALR
Kos, Clair M., Maj. (M.C.), 0356226
School of Aviation Medicine ALR
Matthews, John L., Lt. Col. (M.C.),
School of Aviation Medicine OP
Payne, B. F., Maj. (M.C.), School of Aviation Medicine OP
Reeh, Merrill J.....Lt. Col. (M.C.), Station Hospital OP
Schultz, Jacob F.....Maj. (M.C.), Station HospitalOP
Scobee, R. G., Lt. (M.C.), School of Aviation Medicine
Dept. of Ophthalmology, Research Section OP
Senturia, Ben H., Capt. (M.C.),
School of Aviation Medicine, Research Section ALR
Smith, Harold D., Capt. (M.C.),
School of Aviation Medicine ALR

SAN ANGELO
Windham, Robert Edward.....234 W. Beauregard blvd. OALR

SAN ANTONIO
Atkinson, D. T.....Medical Arts bldg., Z5 OALR
Boehs, Charles John.....Medical Arts bldg., Z5 ALR
Champion, Albert N.....Medical Arts bldg., Z5 ALR
Cranston, William J. Jr., Capt. (M.C.),
Station Hospital, S.A.A.C.C. OP
Dumas, Edward DeVan.....Medical Arts bldg., Z5 ALR
Gill, William Davis.....705 E. Houston, Z5 OALR
Muldoon, Wilfred E.....Nix Professional bldg., Z5 OP
Parrish, Robert E.....Medical Arts bldg., Z5 OALR
Rosebrough, Frank Henry....South Texas Bank bldg., Z5 OP
Russell, Daniel A.....Medical Arts bldg., Z5 ALR
Sykes, Edwin Meredith.....Nix Professional bldg., Z5 ALR
Townes, C. D.
Lt. Col. (M.C.), Station Hospital, S.A.A.C.C. OP

SHEPPARD FIELD
Montgomery, John Lee....Maj. (M.C.), Station Hospital OP
Steffensen, W. H., Capt. (M.C.).....Sta. Hosp. ALR-PI S

SHERMAN
Thomas, W. Maxwell
Lt. Comdr. (M.C.), 437 W. Houston OP
Waltz, Harold D.....Maj. (M.C.), Perrin Field ALR

STEPHENVILLE
Terrell, Vance.....Stephenville Hospital ALR

TAYLOR
Alexander, Margaret Henrietta.....720 Talbot ALR

TEMPLE
Dowling, J. R.
Maj. (M.C.), McCloskey General Hospital ALR
Klawans, Harold L., Capt. (M.C.),
McCloskey General Hospital ALR
McDavitt, Bertha Schenck Stecker.....Main and av. A ALR
Smith, Henry C., Lt. Col. (M.C.), McCloskey Gen. Hosp. OP
Stevenson, Walter D. Jr., Maj. (M.C.),
McCloskey General Hospital ALR
Watkins, J. O., Maj. (M.C.), McCloskey Gen. Hospital OP
Woodson, B. Palmer.....Scott and White Hospital OALR
Woodson, W. Burbank.....601-22 Professional bldg. OALR

TEXARKANA
Fuller, T. E.....209 Pine OALR
Mann, Albert H.....2324 Glendale av. ALR
Roberts, A. Warren.....209 Pine OALR

TYLER
Vaughn, E. H.....Peoples Nat'l Bank bldg. OALR

WACO
Aynesworth, H. T.....Medical Arts bldg. OALR
Burgess, John Louis.....Medical Arts bldg. ALR
Pryor, Will R., Maj. (M.C.), Station Hosp., W.A.A.F. OP
Spencer, Shelby Cruthirds.....Professional bldg. OALR
Woolsey, William Jinkins.....212 N. 7th st. OP

WAXAHACHIE
Donnell, Herbert.....507 W. Main ALR

WICHITA FALLS
Nail, James B.....1300 Eighth ALR

UTAH

BRIGHAM
Keyes, John E. L., Maj. (M.C.), Bushnell Gen. Hospital OP
Lewin, Michael, Capt. (M.C.),
Bushnell Gen. Hospital ALR-PI S

HOLLIDAY
Palmer, Bascom W.....2491 E. Valley View OP

KEARNS
Hunnicutt, Leland G., Maj. (M.C.), Station Hospital ALR

OGDEN
Hetzel, C. C.....Eccles bldg. OALR
Pugmire, Ralph W.....2404 Washington bldg. OP

PROVO
Oaks, Lewis Weston.....33 E. 2nd S. OALR

UTAH — Continued

SALT LAKE CITY

Le Compte, E. D.....9 Exchange place, Z1 OALR
Maw, Raymond B.....699 East South Temple, Z2 ALR
Smith, William Leroy.....Medical Arts bldg., Z1 OP
Stauffer, F. Leaver.....52 East South Temple, Z1 ALR
Welsh, Thomas F.....First Nat'l Bank bldg., Z1 OALR
White, Vivian P.....Tribune Telegram bldg., Z1 ALR

VERMONT

BARRE

Jarvis, De Forest C.....Quarry Bank bldg. ALR

BURLINGTON

Arnold, Francis J.....182 Pearl OALR
Twitchell, Emmus George.....266 Main OALR

MONTPELIER

Bertrand, George Joseph.....73 Main st. OP

RUTLAND

Marshall, George Guerin.....Gryphon bldg. OALR

ST. JOHNSBURY

Cramton, Edward Allen.....33 Main OALR

VIRGINIA

CHARLOTTESVILLE

Burton, Edwin Weisman.....Y.M.C.A. bldg. OP
Krimsky, Joseph.....Rio road OALR
Woodward, Fletcher Drummond.....104 E. Market ALR

COVINGTON

Winn, Thomas Meredith.....129½ Maple av. OALR

DANVILLE

Dickerson, William E., Maj. (M.C.), 802 Masonic Temple ALR
McGovern, Francis H.....Masonic Temple bldg. ALR

FORT BELVOIR

Hayes, Merrill B.....Maj. (M.C.), Station Hospital ALR

FREDERICKSBURG

Holloway, J. Minor.....1200 Prince Edward ALR

HARRISONBURG

Preston, H. Grant.....Professional bldg. ALR

LYNCHBURG

Gorman, James R.....Allied Arts bldg. ALR
Holland, John Gill.....725 Church ALR
Morrison, Frederick M., Lt. Comdr. (M.C.),
Allied Arts bldg. OP

NEWPORT NEWS

Birch, John R., Maj. (M.C.),
Station Hospital, Camp Patrick Henry ALR
Hankins, George Geddy.....Medical Arts bldg. ALR
Poindexter, Frank Wilmore.....Medical Arts bldg. ALR

NORFOLK

Benkwith, Karl B., Lt. (M.C.), Lion Three (G)
Camp Allen, N.O.B. Z11 OP
Campbell, Edward H., Lt. Comdr. (M.C.),
USNR, Naval Hospital ALR
Cooley, Carl Conrad.....Medical Arts bldg., Z10 ALR
Culler, Arthur M., Comdr. (M.C.), 1007 Baldwin av. OP
O'Rourke, Carroll
Lt. Comdr. (M.C.), U. S. Naval Hospital N.O.B., Z11 ALR
Provost, Pierre E.....Lt. (M.C.), Naval Air Station, Z11 ALR
Silcox, Louis E., Lt. (M.C.), U. S. Naval Hospital, NOB ALR
White, J. Warren.....Medical Arts bldg., Z10 OALR

PORTSMOUTH

Smith, Austin T., Comdr. (M.C.), Norfolk Naval Hospital ALR
Smith, Thomas T., Lt. (M.C.), Norfolk Naval Hospital ALR

PULASKI

Smith, Richard O.....73 Third av. N. W. ALR

QUANTICO

Bloch, Charles L., Lt. (M.C.), USNR, Post Dispensary OP
Samponaro, Nicholas, Lt. Comdr. (M.C.), USNR
U. S. Naval Hospital ALR

RICHMOND

Brandwan, Samuel R., Capt. (M.C.),
Station Hospital, Army Air Base OP
Courtney, Robert H.....Professional bldg., Z19 OP
Gatewood, E. T.....Professional bldg., Z19 ALR
Hughes, T. E.....1000 W. Grace, Z20 ALR
Pastore, Peter Nicholas
Medical College of Virginia Hospital, Z19 ALR
Wallerstein, Emanuel U.....Professional bldg., Z19 ALR
Wellford, B. R.....510 Medical Arts bldg., Z19 ALR

ROANOKE

Gill, E. G.....711 S. Jefferson, Z10 ALR
Hamlin, Fred Eugene.....Medical Arts bldg., Z11 ALR
Hatcher, Wm. F.....Medical Arts bldg., Z11 OP
Stone, Harry B.....Medical Arts bldg., Z11 ALR

STAUNTON

Fisher, Guy Rothwell.....1 E. Beverley st. ALR

WILLIAMSBURG

Bell, Julius W.....Lt. Comdr. (M.C.), Camp Peary ALR
Combs, Arnold B.....Lt. (M.C.), USNR, Camp Peary ALR

WINCHESTER

Barlow, Roy A..... ALR
Hawthorne, Allen T.....110 N. Braddock ALR
McGuire, H. H.....105 N. Braddock OP
McGuire, William P., Lt. Comdr. (M.C.),
717 South Stewart st. OP

WASHINGTON

BELLINGHAM

Howe, Spencer S.....Bellingham Nat'l Bank bldg. OALR

BREMERTON

Adix, H. Victor Jr., Lt. Comdr. (M.C.),
U. S. Naval HospitalALR
Jones, Lester T.....Comdr. (M.C.), Naval Base Hospital ALR

COLFAX

Henry, Robert.....Capt. (M.C.), 322 E. James st. OP

ELLENSBURG

Hicks, William Wynne.....202 E. 6th st. ALR

WASHINGTON — Continued

EVERETT

Murphy, Albert B.....Medical and Dental bldg. ALR
Powers, H. W.
Maj. (M.C.), Station Hospital, Paine Field ALR
Secoy, Harry R.....Central bldg. ALR

FORT GEORGE WRIGHT

Hagan, Cornelius, E., Jr., Capt. (M.C.), A.A.B. Hospital ALR
Mullen, C. J.....Maj. (M.C.), A.A.B. Hospital OP

FORT LEWIS

Snyder, Jerome, Lt. (M.C.), 103rd Evacuation Hospital ALR

LONGVIEW

Barton, John F.....1346 Fourteenth av. ALR

PASCO

Flynn, J. Donald
Lt. (M.C.), Naval Air Station Dispensary ALR

SEATTLE

Adams, Frederick W.
Roosevelt Clinic, 1114 Boylston av., Z22 OALR
Bentley, Frederick.....Cobb bldg., Z1 OP
Chase, E. Frank.....Cobb bldg., Z1 OALR
Dowling, J. Thomas.....1115 Terry av., Z1 OALR
Hoffman, W. F.....1317 Marion, Z4 OP
Jensen, Carl Dana, Lt. (M.C.),
Medical and Dental bldg., Z1 OP
Johnson, David M., Lt. Comdr. (M.C.), Naval College
Training Unit, University of Washington, Z5 ALR
McCoy, L. L.....1317 Marion, Z4 OP
Miller, Alvin R.....Fourth and Pike bldg., Z1 ALR
Peden, Barton Edward.....Stimson bldg., Z1 ALR
Shannon, C. W.....New 4th and Pike bldg., Z1 OP
Slack, Walter K., Comdr. (M.C.),
U. S. Naval Hospital, Z4 ALR
Stillson, H.....Seabord bldg., Z1 OALR
Tolan, John F.....1115 Terry, Z1 ALR
Weber, Julius Albert.....Stimson bldg., Z1 ALR

SPOKANE

Epstein, Sidney, Capt. (M.C.)..Baxter General Hospital OP
Hantman, Irvin, Capt. (M.C.),
A.S.C. Spokane Army Air Field ALR
Meyers, Paul Thomas, Maj. (M.C.), 82nd Gen. Hosp. ALR
Parsell, Louis A.....Paulsen Med. and Dental bldg., Z8 ALR
Rott, O. M.....1828 W. 1st av., Apt. C, Z9 OALR
Smith, Carroll L.....Paulsen Med. and Dental bldg., Z8 OALR
Veasey, C. A., Sr.....Paulsen Med. and Dental bldg., Z8 OP
Veasey, C. A., Jr.....Paulsen Med. and Dental bldg., Z8 OALR

TACOMA

Howe, A. W.....Medical Arts bldg., Z2 OALR
Ludwig, William Henry.....Medical Arts bldg., Z2 ALR
Lundvick, Cyril V.....Medical Arts bldg., Z2 OP
Paul, William C.....Medical Arts bldg., Z2 ALR

VANCOUVER

Bradley, John W., Maj. (M.C.), 76th General Hospital ALR
Dunnavan, Floyd Leighton.....Arts bldg. OP
Sugar, H. Saul..Maj. (M.C.), Barnes General Hospital OP
Weisser, Charles William, Capt. (M.C.), 76th Gen. Hosp. OP

WALLA WALLA

Bailey, Paul....Maj. (M.C.), McCaw General Hospital ALR
Ronis, Bernard J., Capt. (M.C.), McCaw General Hospital ALR

WEST VIRGINIA

BECKLEY

Peter, Beverly K.....Beckley Hospital ALR

BLUEFIELD

Blaydes, J. E.....First Nat'l Bank bldg. OP
McGuire, John.....Peery bldg. ALR
St. Clair, C. T.....Cor. Bland and Ramsey ALR

CHARLESTON

Holcombe, Virgil Eugene.....Medical Arts bldg., Z1 ALR
Seletz, Abraham.....Medical Arts bldg., Z1 ALR

CLARKSBURG

Thomas, Harry Vernon.....Empire Bank bldg. OP

ELKINS

Goodwin, Thomas M.....Golden Clinic ALR

FAIRMONT

Johnson, H. R.....320 Jefferson OALR
Maxwell, Joseph Sprague.....Professional bldg. OP
Traugh, George H.....309 Cleveland av. ALR

HUNTINGTON

Beckner, William F.....1050 Fifth av., Z1 ALR
Moore, J. Hallock.....1050 Fifth av., Z1 ALR
Moore, T. W.....1050 Fifth av., Z1 OP

MARTINSBURG

Alexander, Fred William, Maj. (M.C.),
Newton D. Baker General Hospital ALR
Fox, Sidney A., Maj. (M.C.),
Newton D. Baker General Hospital OP

MONTGOMERY

McGehee, Melvin Wood.....Laird Memorial Clinic ALR

MORGANTOWN

Wylie, C. B.....293 High OALR

PARKERSBURG

England, Welch.....1009 Market ALR
Gilmore, Milton A.....Box 172 OP
Hartman, Eugene C.....610½ Market OALR

WELCH

Schiefelbein, Harry T.....Stevens Clinic Hospital OP

WHEELING

Fawcett, Ivan.....75 Twelth st. OALR
Jones, Edmund Lloyd.....Wheeling Clinic OALR
Tomassene, R. A.....1144 Market OALR
Zubak, Matthew F.....Citizens Mutual Trust bldg. OALR

WHITE SULPHUR SPRINGS

Croll, Leo J.....Capt. (M.C.), Ashford General Hospital ALR
Hert, C. B.....Maj. (M.C.), Ashford General Hospital ALR
Masters, John M.
Maj. (M.C.), Ashford General Hospital OP

WISCONSIN

APPLETON

Brooks, E. H.....Zuelke bldg. OALR
Rector, A. E.....Zuelke bldg. OALR

WISCONSIN — Continued

ASHLAND
Grigsby, Roll O.....100 E. Second ALR

BELOIT
Shearer, Harry Arthur.....405 E. Grand av. ALR

EAU CLAIRE
Payne, A. L.....119 S. Barstow OALR
Russell, Sidney Benteen.....Midelfart Clinic OP
Tanner, James Wallace.....131 S. Barstow ALR

FOND DU LAC
Folsom, W. H.....11 N. Main OALR

GREEN BAY
Ford, William W.....109 East Walnut ALR
Nadeau, E. G.....Bellin bldg. OALR

JANESVILLE
Pember, Aubrey H.....500 W. Milwaukee OALR

KENOSHA
Ashley, T. W.....5711 Eighth av. OALR

LA CROSSE
Douglas, Frederick Alexander.....State Bank bldg. OALR
Smith, D. S.....State Bank bldg. OALR

MADISON
Davis, Frederick Allison....224 W. Washington av., Z3 OP
Duehr, Peter Alexander.....224 W. Washington av., Z3 OP
Harned, John W., Maj. (M.C.),
Station Hospital, Truax Field, Z7 ALR
Lynch, Joseph S.
Capt. (M.C.), A.A.F.T.T.C., Truax Field, Z7 OP
Neff, E. Eugene.....224 W. Washington av., Z3 OP
Nesbit, Mark E.....616 1st Nat'l Bank bldg., Z3 OP
Nesbit, Wellwood.....1 S. Pinckney, Z3 ALR
Schubert, Clarence K.....1 S. Pinckney, Z3 OP
Smith, R. B.....224 W. Washington av., Z3 ALR

MANITOWOC
Andrews, M. P.....826 S. 8th OALR
Schneck, Nathan.....815½ S. 8th ALR

MARINETTE
May, J. V.....1703 Main OALR

MARSHFIELD
Copps, Lyman Alden.....Marshfield Clinic OALR

MILWAUKEE
Bach, Edwin Clarence.....324 E. Wisconsin av., Z2 ALR
Blankstein, Samuel S.....606 W. Wisconsin av., Z3 OP
Brook, Jeffrey J.....231 West Wisconsin av., Z3 ALR
Cannon, Harold J.....536 W. Wisconsin av., Z3 ALR
Carl, Evan Elsworth.....606 W. Wisconsin av., Z3 OP
Chapman, Vernon A.....324 E. Wisconsin av., Z2 OALR
Fox, Meyer S.....606 W. Wisconsin av., Z3 ALR
Gordon, J. S.....Bankers bldg., Z2 OALR
Grove, W. E.....324 E. Wisconsin av., Z2 OALR
Haessler, F. Herbert.....Wells bldg., Z2 OP
Heeb, Harry J.....212 W. Wisconsin av., Z3 OALR
Higgins, S. G.....411 E. Mason, Z2 OALR
Hitz, H. B.....411 E. Mason, Z2 ALR
Judge, Thomas Ambrose.....735 N. Water, Z2 ALR
Light, George.....709 N. 11th, Z3 ALR
McCormick, Thomas F.....Wells bldg., Z2 ALR
Martin, Hilmar George.....231 W. Wisconsin av., Z3 OP
Molsberry, Jasper Merl.....425 E. Wisconsin av., Z2 OP
Schmidt, Herbert G.....411 E. Mason, Z2 OALR
Schoofs, Orlando P.....324 E. Wisconsin av., Z2 ALR
Sproule, Ralph P.....208 E. Wisconsin av., Z2 OALR
Tolan, Thomas L.....Wells bldg., Z2 ALR
Treskow, Frank G.....411 E. Mason, Z2 ALR
Waldeck, Edward A.....238 W. Wisconsin av., Z3 OALR
Warner, Raymond C.....324 E. Wisconsin av., Z2 OP

MONROE
Kundert, Fred W.....The Monroe Clinic ALR

OSHKOSH
Ebert, Reinhold O.....64 Mt. Vernon st. OP

RACINE
Beeson, Henry B.....423 Main ALR

SHEBOYGAN
Neumann, William H.....Sheboygan Clinic OALR

STEVENS POINT
Dunn, Austin Gibbons.....111½ Strongs av. ALR

SUPERIOR
Myers, Elmer A.....Board of Trade bldg. OALR

WAUSAU
Trumbo, James Keeley.....526 Third ALR
Wilson, Otis M.....502 3rd st. OP

WHITEHALL
Simons, Neal S.....MacCornack Clinic ALR

WYOMING

CASPER
James, George Russell.....Wyoming Nat'l Bank bldg. OP

CHEYENNE
Newman, Erwin W.....1606 Capitol av. OP
Williams, Russell I.....422 Hynds bldg. ALR

FORT F. E. WARREN
Wilson, Wm. H.....Capt. (M.C.), Station Hospital ALR

SHERIDAN
Veach, Oscar Lloyd.....Whitney Trust bldg. OP

CANADA

ALBERTA

CALGARY
Dowling, Harvey E., F/L R.C.A.F., 414 4th av. West OP

BRITISH COLUMBIA

VANCOUVER
Boucher, R. B.....Medical-Dental bldg. OALR
Brown, W. Harold.....Birks bldg. OALR

VICTORIA
Keys, Morton J.....Pemberton bldg. OALR

MANITOBA

BRANDON

McDiarmid, H. Oliver.....35 Clement block OALR

WINNIPEG

Macneil, Frank A.....Medical Arts bldg. ALR
Ramsey, Robert M.....Flight Lt. R.C.A.F., 320 Yale av. OP

NEW BRUNSWICK

ST. JOHN

Chipman, L. DeV.....42 Coburg OALR
Hayes, Robert T.....42 Coburg ALR

NOVA SCOTIA

HALIFAX

Schwartz, Hugh William.....183 S. Park ALR

ONTARIO

HAMILTON

Macfarlane, P. B.....Medical Arts bldg. OALR
Rabinowitz, Paul.....Mountain Sanitarium ALR

LONDON

McFarlane, Donald C.....380 Queens av. ALR

OTTAWA

Fraser, W. G.....Medical Arts bldg. OALR
Smart, Robert Edwin.....Medical Arts bldg. OP

PETERBOROUGH

Buchanan, Norman D.....129 Murray OALR

TORONTO

Alexander, Seeyman L.....Medical Arts bldg., Z5 ALR
Atkins, John.....345 Bloor st. W. ALR
Barnes, Lancelot.....170 St. George OP
Bochner, Maxwell K.....329 Bloor st. W. OP
Burnham, Howard Hampden.....Medical Arts bldg., Z5 ALR
Campbell, A. A.....86 Bloor st. W., Z5 ALR
Davies, T. A.....578 Sherbourne OALR
Goldsmith, Perry G.....Medical Arts bldg., Z5 ALR
Gollon, Joseph.....425 Bloor st W., Z5 ALR
Hill, C. E.....160 Bloor st W., Z5 OP
Luke, William R. F.....Medical Arts bldg., Z5 OP
McCart, Howard W. D.....170 St. George st., Z5 ALR
McCulloch, Robert J. P.....Medical Arts bldg., Z5 OALR
McEachern, Margaret Aileen.....Medical Arts bldg., Z5 ALR
McGregor, Greger William.....170 St. George, Z5 ALR
McLeod, Angus M., Capt.....Medical Arts bldg., Z5 ALR
Morgan, Alonzo Lloyd.....170 St. George, Z5 OP
Nugent, M. W.
S.L., R.C.A.F., 37 Strathearn road West, Z10 OP
Risdon, E. F.....Medical Arts bldg., Z5 ALR-PI S
Robert, Joseph X.....170 St. George, Z5 ALR
Smith, I. Ruebert.....116 College st. ALR
Snell, George C.....Medical Arts bldg., Z5 ALR
Stark, W. Berkeley.....Medical Arts bldg., Z5 ALR
Sullivan, J. A., Wing Comdr. (RCAF),
Medical Arts bldg., Z5 ALR
Veitch, Ambert H.....170 St. George, Z5 ALR
Wishart, David Edmund Staunton.Medical Arts bldg., Z5 ALR
Wright, Walter Walker.....Medical Arts bldg., Z5 OP

WELLAND

Briant, Thomas Ernest.....28 East Main ALR

QUEBEC

MONTREAL

Ballon, D. H.....1471 Crescent ALR
Freedman, Abraham O.....1396 St. Catherine st. W. ALR
Hodge, George E.....1414 Drummond ALR
Hutchison, Keith O.....1509 Sherbrooke st. W. ALR
Johnston, Kenneth B.....1509 Sherbrooke st. W. OP
Lapointe, Jean.....300 Carre, St. Louis ALR
Lundon, A. E.....Col., 1463 Bishop st., Apt. C-3 ALR
McAuley, Albert G.....1301 Dorchester st. W. OP
McNally, William James.....Linton Apartments ALR
MacMillan, J. A.....1410 Stanley OP
Ramsey, G. Stuart.....1496 Mountain OP
Rogers, J. T.....Medical Arts bldg. ALR
Rosenbaum J.....1396 St. Catherine st. W. OP
Tremble, George E.....1930 Sherbrooke st. W. ALR
Wright, R. Percy, Col. R.C.A.M.C.
6065 Sherbrooke st. W. ALR

EUROPE

ENGLAND

LONDON

Dundas-Grant, Sir James.....(Retired) England
Elliott, R. H., Lt. Col...54 Welbeck st., Cavendish sq. W.
Graves, Basil.11 Devonshire pl., Upper Wimpole st. W., 1
Kirkpatrick, Henry, Lt. Col.....54 Welbeck st. W., 1
Lister, Sir William T.....24 Devonshire pl. W., 1
Parsons, Sir John Herbert
54 Queen Anne st., Cavendish sq. W. OP

GENERAL GOUVERNEMENT

KRAKAV

Lauber, Prof. Dr. Hans.....ul. Gertrudy 18ms OP

HOLLAND

LEIDEN

Van der Hoeve, Prof. J.....Rijnsburgerweg 6A

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